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THE SEX RATIO OF OFFSPRING OF PILOTS OF HIGH PERFORMANCE MILITARY AIRCRAFT

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INTRODUCTION

THERE currently exists in many military fighter units a standard joke among the pilots, which apparently no one except those concerned appears to take seriously, to the effect that fighter pilots are destined to father daughters only. This legend has been universally expressed in fighter squadrons until it has become almost an accepted fact in the minds of many individuals. The purpose of this study has been to investigate the possibility of a correlation between flying military fighter-type aircraft and the sex ratio of offspring of individuals regularly engaged in this type of occupation.

The attention of the author was focused on this question by several related observations. A rather personal interest (and recognized bias) was introduced when the author became the father of two daughters while flying fighter-type aircraft, and since ceasing to fly has had three successive sons. In one fighter squadron in which the author served, 27 female offspring were born to pilots while only 2 males were born during the same period, for a sex ratio of only 7.41. Unfortunately, data are incomplete for this period so no reliable conclusions can be drawn, and these data are not included in this paper. However, it is this type of occurrence that has led to the folklore expressed above, since this sort of skewed sex ratio among offspring of fighter pilots is apparently not just an isolated exception. It appeared possible that such a situation would be masked by the stabilizing figures of the much larger group at any one military base, of which the fighter pilots would be only a small selected segment. For example, in the unit cited above, the extremely lopsided sex ratio of offspring would not be evident in any hospital birth figures since the fighter pilot families comprised less than 1/100th of the

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total base complement. No comparable study has been found either in the literature or through communication with aeromedical sources.

Therefore, it appeared to be of distinct interest to investigate the possibility that the sex ratio of the offspring of military fighter-type pilots may be at a distinct variance with the expected. Military fighter pilots for this study are considered to be those individuals who regularly participate in flight involving psycho-physiological stresses involving high positive and negative gravitational forces, high speed, and usually high altitude flight.

The scope of this investigation has been limited to three separate samples of military fighter pilots. It is felt that these groups are representative of the fighter pilot population as a whole. All three of these groups contain a high ratio of combat-experienced fighter pilots, although the range of experience varies considerably. Two of these groups are operational jet fighter-interceptor squadrons, one in this country and one in Japan. The third, and by far the largest group, consists of all jet fighter instructors at a major training base.

METHODS AND MATERIALS

The primary tool employed in the field work consisted of index cards upon which individual data were noted. These cards contained space for (1) identification by code number of each individual's name, (2) the total number of children, their sex, and birth dates for each and (3) flying time and type of aircraft flown by month prior to conception, or 9, 10, 11, 12 months prior to the birth of each child.

Data were obtained in each instance from two primary sources. The Form 93, or personal and emergency data form, for each pilot individually listed his offspring and birth data for each. The Form 5, kept in the operation section, contains daily, monthly, and yearly breakdowns of flying time for each pilot throughout his flying career, as well as listing each individual flight by aircraft type and mission.

The data for each individual were thus obtained in two steps. First, the personnel folder for each individual had to be examined to obtain the number of children and sex of each, as well as their birth dates. Such facts as whether any offspring were through prior marriage on the part of either wife or husband, since children born to the mother in a prior marriage could not be included, were also noted at this time. Obtaining the birth date for each individual child was essential before the second step could be undertaken, since the flying time desired was

concerned with the four months prior to conception of each child only. Examination of the flight forms for each pilot resulted in obtaining the actual amount of flight time during these periods, as well as the types of aircraft flown, and total experience factors.

The unit names are purposely not given, and the individuals involved were coded, although the author retained a master index. In addition, symbols are used in place of exact flight times. These security considerations have no effect upon the actual data.

The code employed on these forms for type of aircraft was broken down into four major units: F for fighters, M for multi-engined flight, T for trainers, and 0 when no flight time was involved in any specific time period. It should be pointed out that the training-type aircraft were all single-engined, and could be considered to be in the fighter category, since the more recent trainers are quite comparable in speed and maneuverability to World War II fighters. Distinction as to jet or reciprocating engine types was not made because many propeller driven fighters were capable of withstanding comparable g forces to the newer jet series. World War II fighters using full military power at sea level flight could only indicate 300 to 400 knots. To attain speeds equivalent to high dynamic pressure, steep dive angles at low altitude were required, and if anything went wrong the pilot could decrease airspeed by pulling "g's" without fear of causing structural damage to the aircraft. In fact, one manufacturer has made the claim that "no one ever broke the wings" on one type of World War II fighter. In contrast, modern jet fighters are capable of high mach airspeeds in climbs which far exceed the airspeed possible in a World War II fighter in a steep dive at full power. The difference is not so much in the structure, but rather in the relative airspeeds possible.

Further, many of the fighter-interceptor jet types, such as the older F-86D, F-89, and F-94C as well as certain of the "century" series are restricted in maneuverability, and thus in "g" forces experienced, because of the radar and other electronic equipment carried. Therefore, no differentiation was made between propeller versus jet type fighters.

In addition, to account for the wide range of monthly flying time accomplished, minus (—) is used for less than 5 hours per month flight time as a pilot, and plus (+) for over 30 hours in any one month, the latter being considered in general a heavy amount of fighter flying time per month. A symbol with neither sign following it represents an amount between these extremes, or 5 to 30 hours of flying time during the months considered.

DISCUSSION

In most human populations more males than females are born, although some variation has been found between racial groups, nationalities, age groups, and occupations (Scheinfeld, 1944; Strandskov, 1942; and many others). Cultural factors also may have considerable influence upon figures reported from certain groups, particularly in populations where female offspring are held in low esteem. Heath (1954) has claimed that physique and temperament are related to the sex ratio in that mesomorphic and somatotonic individuals tend to have a greater incidence of daughters. Coe (1950) observed that the sex ratio generally ranges from 104 to 107 males per 100 females born.

The sex ratio is defined as the number of males per 100 females. This paper is concerned with the secondary sex ratio, or the proportion of males to females at birth. The primary sex ratio at time of conception, and the tertiary sex ratio, at maturity, differ strikingly in that the primary sex ratio has been shown to be considerably higher (Russell, 1936; Von Hentig, 1952).

The year 1950 is used as a baseline since most of the children who were included in this study were born between 1946-1955. In 1950 there were 1,823,555 male and 1,730,594 female registered births in the U. S. population as a whole (1,863,00 males and 1,768,000 females when adjusted by the census bureau for under registration), which is a sex ratio at birth of 105.37. This figure is used for comparison in this study as the sex ratio at birth for the U. S. population as a whole.

The present study included a total of 236 married pilots, 111 of whom had a total of 222 children. The sex ratio for the pooled sample is 103.67 (113 males, 109 females), or slightly lower than for the U. S. population as a whole.

When this population was categorized according to the type of aircraft the father flew prior to conception, a marked difference is apparent. As is shown in table 1, when the father flew fighters prior to conception, of 94 total children born, 59 were females while only 35 were male offspring. This results in a sex ratio of 59.32, significantly different from the normal expected of 105.37.

These figures are of even greater statistical significance when compared to the offspring which resulted when fathers flew multi-engined types of aircraft prior to conception, and when they did not fly at all prior to conception. In this population 15 females and 25 males were born when the fathers flew multi-engined types of aircraft prior to

TABLE 1

Comparison of the type of aircraft fathers flew prior to conception with resulting sex offspring.

Month of conception (9 months prior to birth of offspring)

CONDITION	GROUP A		GROUP B		GROUP C		TOTAL		RATIO
	fem.	m.	fem.	m.	fem.	m.	fem.	m.	
Flying fighters (F)	30	24	15	10	6	4	51	38	74.51
Flying transports (M)	7	10	6	2	1	4	14	16	114.29
Not flying (0)	25	36	6	3	7	7	38	46	121.05

One month prior to conception (10 months prior to birth)

CONDITION	GROUP A		GROUP B		GROUP C		TOTAL		RATIO
	fem.	m.	fem.	m.	fem.	m.	fem.	m.	
Flying fighters	30	25	15	10	6	4	51	39	76.47
Flying transports	6	10	7	2	1	4	14	16	114.29
Not flying	29	38	5	3	7	7	41	48	117.07

Two months prior to conception (11 months prior to birth)

CONDITION	GROUP A		GROUP B		GROUP C		TOTAL		RATIO
	fem.	m.	fem.	m.	fem.	m.	fem.	m.	
Flying fighters	32	22	15	9	6	4	53	35	66.04
Flying transports	4	15	7	1	1	4	12	20	166.67
Not flying	30	40	5	5	7	7	42	52	123.81

Three months prior to conception (12 months prior to birth)

CONDITION	GROUP A		GROUP B		GROUP C		TOTAL		RATIO
	fem.	m.	fem.	m.	fem.	m.	fem.	m.	
Flying fighters	31	22	15	9	6	4	50	35	70.0
Flying transports	4	13	6	1	1	4	11	18	163.64
Not flying	29	42	6	5	7	8	42	55	130.95

The actual outcome at birth

CONDITION	GROUP A		GROUP B		GROUP C		TOTAL		RATIO		
	fem.	m.	fem.	m.	fem.	m.	fem.	m.		χ^2 *	P
Fying fiighters	37	21	16	10	6	4	59	35	59.32	7.4	<.01
Flying transports	7	19	7	2	1	4	15	25	166.67	2.03	>.10<20
Not flying	23	43	5	3	7	7	35	53	151.42	2.77	>.05<.10

* Chi Square Test for Goodness of Fit. Expected is based upon 105.37 sex ratio at birth of the 1950 census for the U. S. population as a whole.

$$\chi^2 = \frac{(f^o - f^e)^2}{f^e}.$$

conception. This results in a sex ratio of 166.67, significantly greater than expected. A similar effect was found among the pilots who had for one reason or another not engaged in any flight activities for the four months prior to conception. In this latter instance 35 females and 53 males were born, for a sex ratio of 151.42.

This contrast in the sex ratio between the fathers who flew the fighters prior to conception, on the one hand, and those who either did not fly or who flew multi-engined aircraft, on the other hand, is consistent throughout when each squadron is compared. There is only a 2.4% difference between the extremes when each squadron is considered separately in regard to the percentage of females born when the fathers flew fighters prior to conception. This consistency in regard to percentage of female offspring born lends considerable weight to the thesis.

The secondary sex ratios are constantly much lower than would be expected when the fathers flew fighter type aircraft prior to conception. This ratio was found to be 56.75 for Group A, 62.50 for Group B, and 66.67 for Group C. In the pooled sample 62.7% female offspring (59 females and only 35 males) were born. On the other hand, only 37.5% female offspring were born while fathers were flying multi-engined aircraft prior to conception, and only 39.8% females when the fathers were not flying during this period. These figures appear to indicate that not only is the sex ratio affected by the fathers participation in fighter type flying activities, but that the opposite result occurs, that is, many more males than expected are born, when the father either did not fly or flew transport types of aircraft prior to conception.

In the human the secondary sex ratio also appears to vary with the parity, or chronological number of the birth. Normally, there is a continuous drop in the sex ratio at each succeeding pregnancy (Ciocco, 1938; Myers, 1954; Columbo, 1957). The data of this study afforded an opportunity to compare the parity with this expected condition. This normal expectation appears tenable in the instances when the father was either flying transport types of aircraft or not flying at all prior to conception. However, there is a disagreement with normal parity (table 2) when the father flew fighters prior to conception, since there appears to occur a complete reversal of the expected condition. In this latter category the ratio successively increases with each succeeding birth, contrary to the expected.

The null hypothesis is that flying fighter type aircraft does not affect the expected male-female ratio (secondary sex ratio) or live birth of offspring. To test this hypothesis the Chi-Square Test of Goodness of Fit

was utilized to determine whether the observed differences from the expected could be due solely to chance. This test demonstrates the difference between the observed and expected sex ratio as to whether this is a matter of sampling error or reflects an actual statistically significant difference.

TABLE 2

Parity. Chronological order of births of population compiled by type of flying done by father prior to conception.

A. Fathers flew fighters month of conception of offspring (9 months prior to birth.

	(1st births)	(Second)	(Third)	(Fourth)
Male	17	13	7	3
Female	31	14	6	1
Sex Ratio	58.84	92.85	116.67	300.00

B. Fathers flew transport type aircraft or did not fly month of conception of offspring.

	(1st births)	(Second)	(Third)	(Fourth)	(Fifth)	(Sixth)
Male	41	24	5	1	1	0
Female	22	16	8	3	2	1
Sex Ratio	186.36	150.00	62.5	33.3	50.0	.0

Chi square for this population, when the fathers flew fighter aircraft prior to conception, is 7.4, which results (with one degree of freedom) in a probability of $.01 > p > .001$. Thus we can reject the null hypothesis at the .01 level of significance. It may therefore be concluded that frequent participation of the father in high speed, high stress military fighter aircraft flight prior to conception does significantly affect the normal male-female ratio of live birth of offspring. The fact that the reverse affect was noted among the transport pilots and non-flying pilots in this study suggests, unfortunately, that these results may be an artifact of the numbers involved, despite the χ^2 and the fact that the sex ratio for the pooled sample was 103.67. However, it is felt that the data presented are of a nature difficult to obtain so are of value as an indication of a possible situation overlooked by previous investigators through lack of any other study of this nature.

There have been numerous studies which indicate a positive relationship between conditions of high-speed, high-stress flight and specific

physiological effects (for example, Baker and von Halle, 1953; Mathews, 1944; McFarland, 1937; Shettles, 1947; Spindler, 1953; Stewart, 1952; Towner, 1950; and Webster, 1950). Conditions attributed to this type of activity range from aerosinusitis (Bear, 1958) to a complete absence of gastric secretion in the stomach (Suvorov, 1958), and increasingly include such pathologic conditions as colloid cyst of the third ventricle in pilots (Nelson and Haymaker, 1957). In females, studies of flight nurses indicate that initial flight may result in menstrual disturbances, (Doherty, 1945). No attempt will be made here to suggest reasons for the statistically significant sex ratio discrepancy which has been found in this population. These findings indicate a need for further data rather than interpretation at this time. However, it would seem reasonable to consider the possibility that continual high g and high stress type of flight may trigger (or impede) some chemo-physiological reaction particularly effecting the Y-bearing gametes in man, possibly acting through the endocrine system, especially the sex hormones, as has been suggested by Bernstein (1951). Data unfortunately are also lacking to evaluate other possibilities, such as that of a much greater pregnancy wastage among wives of fighter pilots, involving greater numbers of male offspring. It would be of interest in this respect to examine the sex ratio situation in high performance bomber crews, as well as in further fighter units, since if a similar low-ratio is not found in the bomber crews it might indicate that high-speed and high altitude flight are not factors so much as the accumulative effects of high g loads and physio-psychological stress which is inherent in fighter-type flight to a much greater extent than in most other types of flight activity.

SUMMARY

Data were obtained on 236 married pilots representing the total fighter pilot population at three different military airbases. One hundred and eleven of these individuals fathered 222 children, 113 males and 109 females. The data of birth for each child formed a basis for the assumed date of conception for each offspring, and the flight records for each father were examined for the period from 3-0 months prior to assumed conception.

It was found that there was a statistically significant preponderance of female offspring born when fathers flew fighter type aircraft prior to conception $.01 > p > .001$. Of 94 children conceived and born under such conditions, 59 were females, while only 35 were males. This results

in a sex ratio of only 59.3, compared to an expected ratio of 105.37. It was also found that the relationship of parity (birth order) to the sex ratio agreed with the expected when the fathers flew transport types of aircraft or did not fly prior to conception, but that a significant reversal of this relationship occurred when father flew fighters prior to conception of each child.

This study gives statistical support to the hypothesis that the secondary sex ratio is significantly altered toward a preponderance of female offspring when the fathers have participated in high stress fighter-type flying prior to conception.

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EFFECT OF ABO-Rh INTERACTION ON INFANT HEMOGLOBIN [1]

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I AM extremely grateful to the administrative and laboratory directors of the Providence Lying-In Hospital for allowing us to examine the records necessary for this investigation. I have also benefited from discussions with Dr. Herman B. Chase and Dr. Paul F. Fenton. Dr. George Anderson and Dr. Howard Levene read and contributed to an earlier draft of this manuscript.

INTRODUCTION

It has been known for some time that the hemoglobin content in the blood of infants has been subject to several sources of variation (Wolman, 1957). Some of the more important variables are the nutritional status of the mother, altitude of residence above sea level, and the site of infant anatomy from which the blood sample is drawn. The data to be presented here suggest that the infant hemoglobin content in gm/100 ml is strongly affected by ABO-Rh interaction.

SAMPLE AND METHODS

All women entering the Providence Lying-In Hospital, Providence, Rhode Island, are routinely typed for Rh and ABO. Husbands of Rh-negative women are requested to submit to typing and most comply. Children of Rh-negative women are routinely typed and tested for Rh, ABO, hemoglobin level, and other blood measurements.

Records were collected on all live offspring born to white, Rh-negative women beginning with 1950 and ending with 1958. Inclusion of birth record into the sample was based on the availability of seven pieces of

[1] This investigation was supported by a research grant, B-2356, from the National Institute of Neurological Diseases and Blindness, Public Health Service.

information: mother's Rh (negative) and ABO, father's Rh and ABO, and infant's Rh, ABO, and hemoglobin content. The lack of any of this information was cause for rejection of the case from the sample. Sufficient information was gathered on 1091 single-birth infants and 571 couples.

In the present investigation some parental blood types were recorded more than once since they contributed two or more live children to the sample. It will be shown that pregnancy order does not appear to affect the hemoglobin concentration of the infants. Despite the seven requirements of blood types and hemoglobin content, and the duplication of some parental mating combinations, the distribution of ABO and Rh is representative for this lower New England area. This is not too surprising since prior knowledge of blood types or hemoglobin content did not influence the inclusion or omission of any case in this sample.

In Boston, 45 miles to the north of Providence, MacCready and Manin (1951) found the ABO distribution in 120,281 individuals to be: A = 39.7%, B = 10.8%, O = 45.8%, and AB = 3.7%. In the present Providence sample which includes the 1091 infants and the 2182 adult readings, the combined adult and infant distribution was A = 39.96%, B = 10.57%, O = 46.56%, and AB = 2.92%. There were no significant differences noted between the parental and infant ABO distributions. Using the reaction with Anti-D serum, an unpublished study of single ascertainments undertaken in the Providence Lying-In Hospital Blood Bank has resulted in a ratio of 86% positives to 14% negatives (550 typings). The present sample of 1091 father readings involving multiple ascertainment is 86.7 : 13.3.

It is the purpose of this study to consider closely the distributions of blood-group gene frequencies of adults actually involved in the fertile matings. It is the further purpose of this study to examine ABO-Rh interaction in relation to the infant hemoglobin level.

Following the example of previous investigators (Glass, 1949; Cohen, 1960), the Rh-negative women who have Rh-positive husbands were placed into the categories of non-sensitized (NS) and sensitized (S). In addition, Rh-negative women with Rh-negative husbands were placed into a separate category. Thus three divisions involving Rh-negative women are recognized.

Some analysis was done with ABO genotypes. The standard analysis in determining ABO genotypes was followed. Each parent of an OO child was judged to have contributed one of the O genes. In this manner many AO and BO parental genotypes were identified. The

heterozygote parents were separated from the original phenotypic A and B designations. The remaining populations were referred to simply as A? and B?. These populations are to a considerable degree presumptive homozygotes. It was also possible to determine the infant genotype in a large number of cases.

HEMOGLOBIN

Throughout the nine years represented in this sample, the staff of the Providence Lying-In Hospital has utilized the photoelectric-colorimetric method in determining the hemoglobin content. This method is preferred for large-scale hospital routine since the error introduced into the readings is extremely slight (Ham, 1950). The hemoglobin content has been recorded routinely in gm/100 ml.

In this study only one hemoglobin reading was entered for each child and this in all cases was the earliest one done in the laboratory. Furthermore, only readings which were obtained in the first three days were acceptable for the study. It has been shown that mean hemoglobin values tend to remain at the same level for the first three days and thereafter begin to decrease (Ham, 1950; Wintrobe, 1956; Wolman, 1957).

No cases were rejected or included in the sample on the basis of clinical disorder or other abnormalities. Save for the restrictions noted in the blood typing section and mentioned in this section, all eligible cases were included in the sample.

Wintrobe (1956) provides a mean hemoglobin value of 19.5 for newly born infants. Although it is not so indicated, it is assumed that this mean represents infants from sensitized as well as non-sensitized mothers. In the present sample an overall mean value of 19.89 has been obtained (table 1). As would be expected, the children of non-sensitized women had a higher mean value than children of sensitized women. In general the higher hemoglobin readings in the S column of table 1 are attributable to the earlier pregnancies of the sensitized women.

The mean values for the nine sub-populations representing the years covered in the sample did not show any significant deviations from the overall mean value. Consequently no breakdown by years was considered necessary.

The lowest value recorded was 5 gm/100 ml and the highest was 26 gm/100 ml. Wintrobe (1956) reports that values as high as 27 gm /100 ml have been recorded. The low values are also easily admissible.

Thus in all respects the distribution of hemoglobin values does not disagree with those of previously published reports.

Three potential variables were considered and references pertaining to them may be found in Wintrobe (1956) and Wolman (1957). It has been reported in infants that the sexes exhibit no significant difference

TABLE 1

Hemoglobin readings of 1091 infants

HEMOGLOBIN GRAMS/100 ML	NUMBER NS	NUMBER S	HEMOGLOBIN GRAMS/100 ML	NUMBER NS	NUMBER S
5	0	3	16	28	8
6	0	0	17	71	7
7	0	0	18	115	9
8	0	1	19	145	9
9	0	1	20	225	8
10	2	4	21	136	3
11	3	4	22	41	0
12	0	5	23	86	4
13	4	5	24	37	0
14	9	6	25	90	3
15	12	4	26	2	1

	NUMBER	AVERAGE HEMOGLOBIN GRAMS/100 ML
Non-Sensitized	1006	20.20
Sensitized	85	16.24
Total	1091	19.89

in hemoglobin values. In the present study this was confirmed. Also variations in altitude affect the hemoglobin content, but this does not appear to be of significance in this geographical area. Lastly, the hemoglobin values have been routinely determined by a heel sample. This avoids hemoglobin variations based on different anatomical sites.

ABO DISTRIBUTIONS OF BREEDING POPULATIONS

In a recent paper Cohen (1960) has shown that the ABO distributions within the NS series and within the S series differ significantly from one another and diverge in opposite directions from a theoretical Hardy-Weinberg formulation. There was shown to exist a marked deficiency of ABO-incompatible matings in the Rh-sensitized series and some excess

of ABO-incompatibles in the Rh-nonsensitized series. Thus a relatively larger percentage of ABO-compatible matings become Rh-sensitized whereas a smaller proportion of the ABO-incompatible matings become Rh sensitized. These deviations from expectancy are called "preferential shift" (Cohen and Glass, 1959; Cohen, 1960).

TABLE 2

Distribution of ABO mating types within Non-Sensitized (NS) series

		FATHER'S GROUP				TOTAL	%
		A	B	O	AB		
Mother's	A	150	28	148	9	335	38.91
	B	39	10	41	1	91	10.57
Group	O	157	57	189	18	421	48.90
	AB	2	0	12	0	14	1.63
Total		348	95	390	28	861	
%		40.42	11.03	45.30	3.25		

	NO.	%
Compatible Matings	552	64.11
Incompatible Matings	309	35.89

TABLE 3

Distribution of ABO mating types within Sensitized (S) series

		FATHER'S GROUP				TOTAL	%
		A	B	O	AB		
Mother's	A	20	4	14	2	40	47.06
	B	3	2	4	0	9	10.59
Group	O	9	2	20	0	31	36.47
	AB	2	1	2	0	5	5.88
Total		34	9	40	2	85	
%		40.00	10.59	47.06	2.35		

	NO.	%
Compatible Matings	65	76.47
Incompatible Matings	20	23.53

Results, very similar to those presented by Cohen (1960), have been obtained in the present investigation. Out of the 1091 matings, 732 or 67.1% involve ABO-compatible matings and 359 or 32.9% matings are ABO-incompatible matings. However, after this "breeding population" is broken down into its three component parts, it can be seen that the NS

series shows an elevation in incompatible matings and a concomitant depression in the compatible matings. The reverse trends are true for both of the other series (tables 2, 3, 4, and 5).

TABLE 4

Distribution of ABO mating types within Rh-negative father series

		FATHER'S GROUP				TOTAL	%
		A	B	O	AB		
Mother's	A	21	9	48	0	78	53.79
	B	3	0	13	0	16	11.03
Group	O	13	4	24	1	42	28.97
	AB	4	0	3	2	9	6.20
Total		41	13	88	3	145	
%		28.28	8.97	60.69	2.07		

	NO.	%
Compatible Matings	115	79.31
Incompatible Matings	30	20.69

TABLE 5

Comparisons involving parental ABO distributions

POPULATION	d. f.	χ^2	P
Between parental groups (table 2)	3	6.185	$.20 > P > .10$
Between parental groups (table 4)	3	31.090	$< .001$
Among mothers—all series	6	33.855	$< .001$
Among fathers—all series	6	12.19	$.10 > P > .05$

In the NS series there is an excess of O mothers and deficiencies in A and AB mothers. The fathers on the other hand exhibit excesses in A and AB and deficiencies in O. In the S series stronger trends of divergence between the population of mothers and that of the fathers are observed. Although a χ^2 test wherein two out of eight cells have an expectancy of lower than five may not constitute a rigorous test, there is little reason to doubt that fundamental biological processes are a major cause of this divergence. These findings in the S series parallel exactly the deficiencies and excesses which were noted by Cohen (1960).

The divergence between the parental ABO distributions within the Rh-negative $\times$ Rh-negative series is unquestionably the most severe of the three series. The distributions are so contorted that very near four out of five matings are doubly compatible.

The parental distributions of the three series are so divergent that a logical question to ask is whether their distributions could be random deviations from a common population base rather than divergences of real biological consequence. On the null hypothesis it is observed that the probability is slight that all three mothers' distributions came from the same population. Similar results bordering on the 5% level of significance are obtained for the three father distributions (table 5).

ABO-Rh phenotypic interaction

In a recent review Levine (1958) ably summarizes numerous studies which support his contention that ABO incompatibility noticeably reduces the incidence of Rh hemolytic disease. On the basis of the present study it is now possible to examine the effects of ABO-Rh interaction on infant hemoglobin levels.

TABLE 6

Hemoglobin content according to pregnancy order

PREGNANCY ORDER	NUMBER	BELOW 16 GM/100 ML		16 GM/100 ML AND ABOVE	
		Obs.	Exp.	Obs.	Exp.
1	265	8	7.90	257	257.10
2	331	10	9.87	321	321.13
3	213	7	6.35	206	206.65
4	113	2		111	
5–6	70	2	5.87	68	191.13
7–12	14	1		13	
Total	1006	30	29.99	976	976.01

Pregnancy orders used—1, 2, 3, 4 and more

$\chi^2 = .131$ d. f. $= 3$ $P > .95$

The range of hemoglobin values, 5 gm/100 ml to 26 gm/100 ml, may be divided into two equal parts of 11 units each. The 5-15 segment contains those readings which are most frequently associated with infants having abnormal conditions at birth. Wolman (1957) cites 16 gm/100 ml as the lowest value in the optimal range for infant hemoglobin. Thus the division of the observed range into two parts is meaningful in terms of the hemoglobin level and also useful for statistical analysis since it represents the midpoint in the overall range.

The lower group contains 5.77% of all readings encountered in this study whereas the higher group contains the other 94.23%.

TABLE 7

Eleven representative reproductive histories showing blood types and hemoglobin levels in gm/ml

MOTHER		FATHER		RECORDS FOR LIVE CHILDREN BY PREGNANCY ORDER																												
				1			2			3			4			5			6			7			8			9		
Rh	ABO	Rh	ABO	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H	Rh	ABO	H
—	O	+	O	—	O	17	?	?	?	?	?	?	—	O	21	—	O	17														
—	O	+	B	a	a	a	+	B	16	+	B	21	—	O	21	—	O	25														
—	O	+	A	—	A	20	a	a	a	a	a	a	+	O	23	—	A	18	+	A	20											
—	A	+	A	—	O	20	—	A	16	+	A	18	—	A	17	+	A	19	+	O	13											
—	B	+	O	?	?	?	a	a	a	?	?	?	+	O	17	+	O	16	+	O	22											
—	B	+	B	—	O	19	—	B	17	—	B	16	a	a	a	—	B	21	+	B	18											
—	A	+	O	a	a	a	?	?	?	+	O	19	?	?	?	+	O	23	?	?	?	—	O	17								
—	A	+	A	?	?	?	—	A	18	+	A	20	+	A	19	+	A	19	+	A	24	+	A	17								
—	A	+	A	?	?	?	?	?	?	?	?	?	?	?	?	?	?	?	a	a	a	a	a	a	+	A	22					
—	O	+	A	a	a	a	?	?	?	a	a	a	?	?	?	a	a	a	?	?	?	+	O	19	+	A	19					
—	A	+	B	?	?	?	?	?	?	?	?	?	?	?	?	+	B	20	+	O	19	—	O	18	+	B	25	+	AB	23		

Symbols

? Live child but no further information indicated on hospital records.
a Abortion.

In the remainder of this report, the sensitized series will be omitted. The reason for this is simple. It would be well to first establish the shifts in infant hemoglobin level attributable to ABO-Rh interaction without the complicating features of maternal sensitization, anti-Rh titer values, erythroblastosis, and the like. The relation of the ABO-Rh interaction to the sensitized maternal environment will be the subject of a further report.

Pregnancy order, which is important for so many other attributes concerning human birth, does not appear to affect the hemoglobin levels of the infant (table 6). This also is confirmed in some examples of the longer and more complete reproductive histories gathered in the study (table 7). One reproductive history of 12 pregnancies, involving a mating of Rh — × Rh —, was in the study but not included in table 7 because it would have extended the table unnecessarily. However, a hemoglobin level of 23 gm/100 ml was recorded for this mother's ninth live child (twelfth pregnancy) which followed three prior abortions.

Therefore, the information in tables 6 and 7, lead us to conclude that pregnancy order and prior maternal abortions have no visible effect on the infant hemoglobin level. There is also preliminary evidence that prior maternal stillbirths do not affect the level.

Levine and others have used the interaction of the two blood systems in relation to deficiencies or excesses of live children. In a like manner it is now possible to show the interactions of blood systems in relation to the low and high groups of hemoglobin readings (table 8). The analysis for the complete range was accomplished with the two-tailed χ^2 test. The null hypothesis utilized is that the two systems are essentially independent. It can be readily observed that marked deviations from expectancies have taken place. The Classes I and IV, or no incompatibility and double incompatibility, exhibit excesses of live children while Classes II and III of single incompatibilities show deficiencies.

There are two "Expecteds" provided in the table for the partial ranges. Expected$_1$ is based on the assumption that the two systems are essentially independent. Thus the Expected percentages for the complete range and Expected$_1$ percentages for the partial ranges are identical. The data available show a strong divergence between the lower range and the upper range. The divergence is complementary since the lower range has excesses in Classes II and III and deficiencies in Classes I and IV whereas the upper range has excesses in Classes I and IV and deficiencies in Classes II and III. There are sufficient numbers in the upper range to undertake a χ^2 test and this indicates considerable significance. The

lower range has insufficient numbers in some of its cells but the biological trends of deficiencies and excesses is without question.

Expected$_2$ values in the partial ranges are represented by the observed values of the complete range. It is assumed that the percentages of observed values ought to be obtained in any segment of its overall range. Here again the divergence of the two partial ranges are noted and the trends are clear.

TABLE 8

Relationship of ABO-Rh interaction to high and low hemoglobin readings

HEMOGLOBIN RANGE	MATINGS I ABO Comp. Rh Comp.	II ABO Incomp. Rh Comp.	III ABO Comp. Rh Incomp.	IV ABO Incomp. Rh Incomp.	χ^2	P
5–26 (Obs. No.)	115	30	552	309	12.157	< .001
5–26 (Exp. No.)	96.14	48.86	570.86	290.14		
5–26 (Obs. %)	11.43	2.98	54.87	30.72		
5–26 (Exp. %)	9.55	4.86	56.75	28.84		
5–15 (Obs. No.)	0	2	24	4		
5–15 (Exp.$_1$ No.)	2.87	1.46	17.03	8.65		
16–26 (Obs. No.)	115	28	528	305	15.47	< .001
16–26 (Exp.$_1$ No.)	93.20	47.43	553.88	281.48		
5–15 (Obs. No.)	0	2	24	4		
5–15 (Exp.$_2$ No.)	3.43	.89	16.46	9.22		
16–26 (Obs. No.)	115	28	528	305	.255	.70 > P > .50
16–26 (Exp.$_2$ No.)	111.56	29.09	535.53	299.83		

Yet a third Expected could be developed. This would involve the assumption that the observed end totals of each partial range are randomly distributed throughout the four cells. Here the Fisher exact probability test could be applied to the lower range and statistical significance recorded for it. The upper range would exhibit significance by the standard two-tail χ^2 test. The other two Expecteds are more biologically meaningful in this situation because they involve the expectancy that the systems are independent and secondly that the observed values for the complete range ought to apply equally well to all segments of its range.

It is obvious that the hemoglobin content in the liveborn infant is a good index of prior intra-uterine stress.

ABO-Rh GENOTYPIC INTERACTION

There are sufficient cases of the infant group, AO Rh + Rh —, to discuss them on a genotypic basis. First, however, it will be essential to provide data which discounts any direct relationship between parental blood type and infant hemoglobin (table 9). There were no real differences in the distribution of infant hemoglobin levels between the NS series and the negative × negative series so these two series are combined in the table.

TABLE 9

Relation of parental ABO genotypes to infant hemoglobin

GENOTYPING	NO. READINGS	BELOW 16GM/100 ML		16GM/100 ML AND ABOVE	
		No.	%	No.	%
	MOTHERS				
AO	192	8	4.17	184	95.83
A?	211	5	2.36	206	97.63
OO	463	14	3.02	449	96.98
	FATHERS				
AO	192	3	1.56	189	98.44
A?	197	7	3.55	190	96.45
OO	478	18	3.77	460	96.23
	COMBINED MOTHERS AND FATHERS				
AO	348	11	2.86	373	97.14
A?	408	12	2.94	396	97.06
OO	941	32	3.40	909	96.60

The AO Rh + Rh — infants exhibit two different distributions of hemoglobin readings and these are based upon parental matings (table 10). The two infant populations resulting from ABO-Compatible/Rh-Incompatible matings have a significant excess of lower hemoglobin readings when contrasted to the two populations resulting from matings of double incompatibilities.

DISCUSSION

The present sample has an ABO distribution which is representative for this area. This population has already produced over 1000 live-born infants. The F_1 generation shows an ABO distribution which is essentially similar to the P_1 generation. However this duplication of the

TABLE 10

Relation of parental matings to hemoglobin content of 173 AO Rh + Rh — infants

MATINGS		BELOW 16 GM/100 ML	16 GM/100 ML AND ABOVE	
ABO ♀ × ♂	Rh ♀ × ♂	No.	No.	Total
AO × OO	− × +	5	29	34
A? × OO	− × +	5	49	54
	Total	10	78	88
OO × AO	− × +	0	42	42
OO × A?	− × +	1	42	43
	Total	1	84	85
	Totals	11	162	173

$\chi^2 = 5.9$ d. f. = 1 $P < .02$

parental population has been accomplished by means of a summation or balance of some very diverse reproduction mating patterns.

Within each of the series, non-sensitized Rh-negative women × Rh-positive husbands, sensitized Rh-negative women × Rh-positive husbands, and Rh-negative women × Rh-negative husbands, there was a considerable difference between the parental ABO distribution. The three maternal groups deviate considerably from each other. Similarly, deviations are noted among the three paternal groups. The matings between sensitized women mated to Rh + husbands contained excesses of ABO-compatible matings and deficiencies in the ABO-incompatible matings. Rh + men mated with non-sensitized women resulted in deficiencies of ABO-compatible matings and excesses in ABO-incompatible matings.

It has been suggested previously that the excesses of fertile matings in the double incompatibility situations when contrasted to the Rh-incompatible/ABO-compatible matings are best explained by the protective effect of the ABO incompatibility on the incompatible Rh mating (Levine, 1958; Cohen, 1960).

In the series containing Rh-negative fathers, the ABO distribution is characterized by a significant lack of incompatible matings. Yet when these three "atypical" series are brought together, the deficiencies and excesses in ABO distributions cancel one another.

The genic interaction of the two systems affects the number of children born to fertile parents. The distribution is not on a random basis. The two classes of no incompatibility and double incompatibility (Classes I and IV) show disproportionately more children than the

number of children in the classes of single incompatibilities between parental matings (Classes II and III).

Another facet of this genetic interaction is its effects upon hemoglobin level of new born infants. Once again the distribution is not random, that is, if it is assumed that the system ought to act independently. In the higher group (16-26 gm/100 ml), Classes I and IV show excesses while Classes II and III have deficiencies. In the lower group (5-15 gm/100 ml), the reverse is true and the deficiencies are now in Classes I and IV and the excesses are in Classes II and III.

One other piece of information is unfortunately not available. This is the effect of ABO-Rh interactions on questions of sterility. It would be a good guess that single incompatibility matings provide a disproportionate number of sterile matings in contrast to the matings involving no and double incompatibilities. No investigation seems to have been done along these lines.

The results and comments of this study and previous studies which bear on ABO-Rh interaction may be presented in the following form.

Condition	*Classes I and IV*	*Classes II and III*
Sterile mating.	lesser chance *	greater chance *
Fertile mating.	greater chance	lesser chance
Elimination of ABO incompatibility zygote.	lesser chance	greater chance
Larger number of children.	greater chance	lesser chance
Normal infant hemoglobin reading.	greater chance	lesser chance
Abnormal infant hemoglobin reading.	lesser chance	greater chance

* an assumption

Hence some parental genic combinations appear to be favored while others are not. The evidence indicates a sort of "continuum of reproductive deficiency and abnormality" for a population of unfavorable genetic matings. The continuum stretches from the time of fertilization past the time of birth into the post-natal period. It would, of course, be highly desirable to know whether these unfavorable genic combinations have long term residual effects in childhood and adulthood.

SUMMARY

There are two purposes to this report. The first being to ascertain the parental ABO distribution in three types of matings:

♀ ♀ Rh — × ♂ ♂ Rh —, Sensitized (S) ♀ ♀ Rh — × ♂ ♂ Rh +,
and Non-Sensitized (NS) ♀ ♀ Rh — × ♂ ♂ Rh +.

The second was to seek the relation of ABO-Rh interaction to infant hemoglobin in the NS and Rh-negative father series.

1. The parental and filial populations show similar ABO distributions and both are typical for the area. The filial distribution represents the consequence of the three very "atypical" ABO mating distributions. Considerable excess of ABO-compatible matings were found in the Rh-negative × Rh-negative series and the sensitized series. An excess of ABO incompatibility was found in the non-sensitized series.

2. Matings of persons compatible in both systems (ABO compatible /Rh compatible) or with double incompatibilities (ABO incompatible /Rh incompatible) tend to show higher infant hemoglobin values than matings involving single incompatibilities (ABO compatible/Rh incompatible and ABO incompatible/Rh compatible).

3. Unfavorable parental genic combinations (single incompatibilities) are quite likely to show the effects of intra-uterine selection and stress from zygote to the post-natal period. This is called a continuum of reproductive deficiencies and abnormalities and the details are discussed.

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THE BLOOD GROUPS OF NATIVES IN KAINANTU, NEW GUINEA

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THE native peoples of the island of New Guinea present an admirable opportunity for the study of human variation in its several forms. Numbering overall between two and three million, they are broken into some 400 language groups—a truly astonishing development of local differentiation and specialization. New Guinea peoples, considering they inhabit a single—though very large—island, display relatively great cultural diversity as well. It could thus be expected that studies of the human genetics of the area might discover a somewhat parallel situation. Indeed, the *kuru* pathology (Zigas and Gajdusek, 1957), now considered to be genetic in origin (Dobzhansky, 1960), is highly localized in one language group, the Fore. This appears to be dramatic evidence of an extremely high degree of genetic specialization. In fine, because of the presence of a large series of variant human communities and populations, New Guinea offers an unusually advantageous situation in which to study gene flow and other problems of population dynamics. Against such a background several New Guinea localities were investigated in 1959 to determine a possible setting for a large-scale study of this kind. In one locality the blood samples were collected upon which the present paper is based.

On behalf of the Committee on New Guinea Studies—a group of anthropologists from six Pacific Coast universities of Canada and the United States of America (British Columbia, Washington, Oregon, California, Stanford and U. C. L. A.), one of the authors (J. B. W.), with the support of a National Science Foundation grant, recently visited the Kainantu region of the New Guinea Highlands (figure 1). In collaboration with V. Z., he collected blood samples from 375 native subjects and sent to Sydney, Australia, where the blood groups were determined.

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Kainantu is an administrative subdistrict of the Eastern Highlands District of Australian New Guinea. Most of the area inhabited by the subjects studied lies between 5,000 and perhaps 6,500 feet above sea level. The Kainantu station, first established in the early thirties, is the

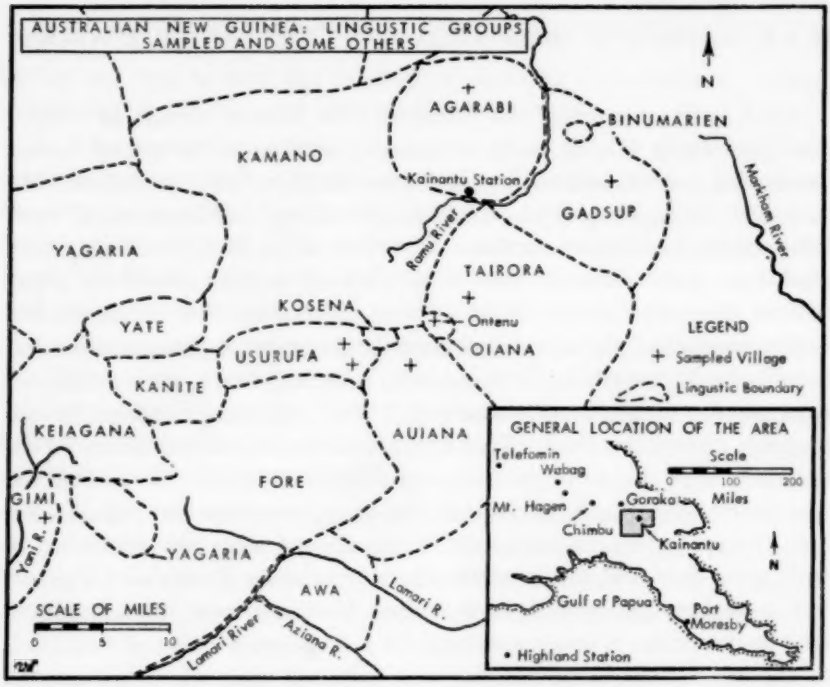

FIG. 1. AUSTRALIAN NEW GUINEA: LINGUISTIC GROUPS SAMPLED AND SOME OTHERS.

location of the principal administrative post in the subdistrict, as well as an airstrip, wireless station, two missions, a government school, and several small commercial establishments. Two great river systems drain the area through mountain ranges to the sea, the Upper Ramu toward the east and north, the Purari on the west and south. While the mean size of language groups (it is misleading in New Guinea to equate them with "tribes") of the Kainantu area is greater than in typical coastal areas, it is the smallest in the highlands, so far as is known. Indeed, there appears to be a fairly consistent east-to-west trend in this respect along the highland backbone of the island—at least in Australian territory—and Kainantu is the easternmost segment.

The purpose of this report is to present the blood group gene frequencies found in the area. They are of some interest because a variant of the Rh genotypes has been found.

MATERIALS AND METHODS

A blood sample was collected from a cubital vein of each subject and placed in a small vaccine vial containing sodium versenate, penicillin and streptomycin, as described by Macintosh, Walsh and Kooptzoff (1958). The samples were sent by air to Sydney and were tested not more than eight days after collection. Tests were performed on saline suspensions of twice washed red cells.

RESULTS

The ABO groups have been analyzed as shown in table 1. All samples which reacted with anti-A were also tested with anti-A_1, but no examples

TABLE 1

ABO blood groups in Kainantu

GROUP	OBSERVED ABSOLUTE	OBSERVED FREQUENCY	EXPECTED ABSOLUTE
O	166	0.4427	163.40
A_1	130	0.3467	127.99
B	65	0.1733	63.99
A_1B	14	0.0373	19.69
Total	375	1.0000	375.07

Gene frequencies: $O = 0.6601$
$A_1 = 0.2214$
$B = 0.1186$
1.0001

$\chi^2 = 1.24$ for 1 degree of freedom,
$0.30 > P > 0.20$, not significant.

of the A_2 group were found. The high frequency of reactions with anti-B (21%) and the absence of group A_2 is characteristic of New Guinea natives. The gene frequencies were calculated by Fisher's method, described by Allison, Mourant, Ikin and Raper (1952).

The MNS groups are shown in table 2. In calculating the gene frequencies it was assumed that S is associated only with N. The frequencies of M and N were calculated by standard methods and the value of S (obtained from $\bar{S} = 1 - \sqrt{\overline{MsMs} + \overline{MsNs} + \overline{NsNs}}$) taken as the frequency of the NS gene. The low frequency of M is characteristic of the highlands of New Guinea but the percentage of positive reactors with anti-S (8.54) is somewhat lowers than that found in adjacent regions.

TABLE 2

MNS blood groups in Kainantu

GROUP	OBSERVED ABSOLUTE	OBSERVED FREQUENCY	EXPECTED ABSOLUTE
MM.S	0	0.0000	0.00
MsMs	1	0.0027	1.54
MN.S	1	0.0027	2.10
MsNs	45	0.1200	42.83
NN.S	31	0.0827	29.96
NsNs	297	0.7920	298.57
Total	375	1.0001	375.00

Gene frequencies: MS = 0.0000
Ms = 0.0640
NS = 0.0437
Ns = 0.8923
1.0000

$\chi^2 = 0.92$ for 2 degrees of freedom,
$0.70 > P > 0.50$, not significant.

The Rh gene frequencies were calculated as follows:

CDe or $CD^ue = \sqrt{(+----)}$

CDe from the quadratic equation

$$(CDe)^2 + 2(CDe)\sqrt{(+----)} = (+-+-)$$

cDe = (+++−) divided by 2(CDe + Cde)

cDE = 1 — (sum of other frequencies)

where the combinations of the plus and minus signs represent the proportions of samples reacting with anti-C, anti-c, anti-D and anti-E respectively, as shown in table 3. The frequency of cDe possibly represents the combined frequencies of cDe and cde, but there is no evidence

to date that cde exists in New Guinea because the homozygote cde/cde has not been found. On the other hand, cDe/cDe was found once in this series and has been detected rarely in other parts of the Territory.

TABLE 3

Rh blood groups in Kainantu

REACTIONS WITH ANTISERA C O D E	MOST COMMON GENOTYPE	OBSERVED ABSOLUTE	OBSERVED FREQUENCY	EXPECTED ABSOLUTE
+ − + −	CDe/CDe	301	0.8027	301.05
+ + + +	CDe/cDE	46	0.1227	45.09
+ − − −	CD^{u}e/CD^{u}e	11	0.0293	10.99
+ + + −	CDe/cDe	15	0.0400	14.98
− + + −	cDe/cDe	1	0.0027	0.18
− + + +	cDE/cDE	1	0.0027	2.71
Total		375	1.0001	375.00

Gene frequencies: CDe = 0.7410
cDE = 0.0659
CD^{u}e = 0.1712
cDe = 0.0219
1.0000

$\chi^2 = 4.83$ for 2 degrees of freedom,
$0.10 > P > 0.05$, not significant.

Eleven subjects gave the reactions of Cde/Cde on initial testing. Further tests were performed as follows:

(a) Negative reactions were obtained when all 11 samples were tested with four complete and eight incomplete anti-D sera of high titre, by the saline, indirect Coombs and P. V. P. (Ward, 1953) methods. This made it unlikely that the samples contained the conventional D^u variant of the D antigen but did not eliminate the possibility that very small amounts of D might be present, *i. e.*, a still "lower grade" of D^u. On further testing a positive indirect Coombs test was obtained with an anti-D serum supplied by R. T. Simmons of Melbourne and also with one from Sydney. It is therefore probable that these subjects have a variant of D which reacts differently to the classical D^u variant but in the remainder of this paper it will be termed "D^u."

(b) Second samples were obtained from ten of the 11 subjects with the variant genotype and findings were confirmed in every instance. Samples were also obtained from ten close relatives, either parents or offspring, of these subjects. The same Rh genotype was found in the father of one of the propositors, as shown in figure 2, but in no other subject.

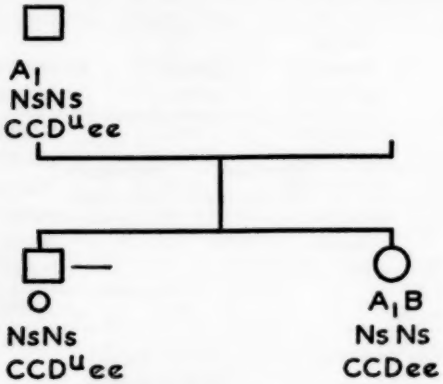

FIG. 2. FAMILY TREE SHOWING SIMILAR RH GENOTYPE IN THE FATHER OF THE PROSPOSITUS.

(c) At the suggestion of Dr. A. E. Mourant of the Lister Institute, London, and with serum kindly supplied by Dr. Phillip Sturgeon of the Children's Hospital, Los Angeles, four of the CDᵘe/CDᵘe propositors were tested with anti-C^N serum. Three failed to react, so that the gene in these subjects is similar to the C^n described by Sturgeon, Fisk, Wintler and Chernock (1958) in Negroes. The anti-C^N serum has also been used against 36 samples from Telefolmin in the western part of Australian New Guinea, all of which had reacted with anti-C and anti-D. There were five negative reactors with anti-C^N, of which two gave the reactions of CDe/CDe, two those of CDe/cDe and one those of CDe/cDE.

All subjects failing to react with the saline anti-D must be homozygous for C, as all gave negative reactions with anti-c, and are either CDᵘe/CDᵘe or C^{n}Dᵘe/C^{n}Dᵘe. It follows that the heterozygotes must be common in this part of New Guinea. From the figures in table 3 it can be calculated that the expected number of such heterozygotes in the series of 375 subjects would be 106 (28.4%).

DISCUSSION

Some blood group gene frequencies in the New Guinea Highlands are listed in table 4, the location of the areas being shown in figure 1. The Kainantu subjects as a group have the same general gene pattern as the natives in adjacent areas and the small differences probably result from such factors as genetic drift, migratory movements and marriage between

TABLE 4

Blood group gene frequencies of some New Guinea Highland peoples

GENE	AREA				
	Wabag [1]	Mt. Hagen [2]	Chimbu [3]	Goroka [4]	Kainantu [5]
A	0.2398	0.205	0.2473	0.1945	0.2214
B	0.1197	0.178	0.1656	0.0934	0.1186
M	0.0528	0.031	0.0249	0.0575	0.0640
S	0.2065	0.177	0.1487	0.0861	0.0437
CDe (R_1)	0.9317	0.954	0.8950	0.8380	0.7410
cDE (R_2)	0.0484	0.034	0.0791	0.1044	0.0659
cDe (R_0)	0.0199	0.012	0.0259	0.0576	0.0219
CD^ue	0	0	0	0	0.1712

Figures taken from:

[1] Macintosh, Walsh and Kooptzoff (1958)
[2] Sample, Simmons, Graydon, Randmae and Jamieson (1956)
[3] Ivinskis, Kooptzoff, Walsh and Dunn (1956)
[4] Kariks, Kooptzoff, Steed, Cotter and Walsh (1959)
[5] This series.

TABLE 5

Glotto-chronological data on languages represented in Kainantu blood series (in roughly west-to-east order)

Gimi	Usurufa	Auiana	Oiana	Tairora	Agarabi	Gadsup
Gimi	—	2400–2800	2550–3050	3450–4050	2450–2950	2450–2950
Usurufa		720–880	1050–1300	—	—	—
Auiana			1350–1650	2350–2750	1500–1800	1650–1950
Oiana (Ontenu)				2300–2700	870–1070	780–920
Tairora					2200–2600	2250–2650
Agarabi						780–920
Gadsup						

Source: S. A. Wurm, Unpublished Ms., 1959, and personal correspondence, 1960.

Figures represent " time-depth," or number of years separating any given pair of languages from their putative common origin. Dashes mean figures not available.

adjacent groups (Macintosh *et al.*, 1958). The decreasing frequency of S from west to east suggests a population movement in either direction along the highland ridge. Within the Kainantu series the west-to-east cline is also rather consistently borne out, although the samples from individual linguistic groups are obviously not large (table 6). The westernmost linguisic group sampled, Gimi, showed 28% of positive reactors, while the two easternmost showed 0% and 3.6% respectively.

TABLE 6

Frequency of positive reactors to anti-S according to language group (groups in roughly west-to-east order)

	TOTAL SAMPLE	NO. HOMOZYGOTES	FREQUENCY
Gimi	25	7	28.0%
Usurufa	38 + 23 = 61 *	5	8.3%
Auiana	45	2	4.4%
Oiana (Ontenu)	64	3	4.7%
Tairora	70	13	18.6%
Agarabi	54	0	0.0%
Gadsup	56	2	3.6%
Total	375	32	8.5%

* Two Usurufa villages were sampled.

Only the Tairora are "out of geographical line"—markedly so, but linguistic and ethnological evidence concerning the position of these people is not inconsistent with such a "discrepancy."

The C gene. Several allelic variants at the C locus have been described, including C, c, C^w, C^x, c^v and C^u. More recently Sturgeon *et al.* (1958) found a serum (No. 127) which gave almost concordant results with anti-C sera against blood samples from Caucasians but not against samples from Negroes. They found that 42 of 124 Negroes (33.9%) giving positive reactions with anti-C failed to react with serum No. 127. The antigen of these 42 samples was termed C^n and the serum No. 127 anti-C^N, a situation which is analogous to the way anti-A_1 delineates A_2 (Sturgeon, personal communication). Sturgeon and his co-workers found that all 20 Negroes with the phenotype Cde were negative with anti-C^N. Some evidence was obtained that C^n may be identical with C (described by Allen and Tippett, 1958), with c^v (reported by Race, Sanger and Lawler, 1948; Arnold and Walsh, 1951), and with Ce (rh_1) of Rosenfield and Haber (1958).

The finding of C^n in New Guinea natives cannot be interpreted as suggesting an ethnic relationship between the New Guinea natives and the Negroes, but it does increase the anthropological value of the anti-C^N serum.

The Rh Chromosome. It is of interest that the combined frequency of CDe and CD^ue in the Kainantu region is approxmiately the same as the frequency of CDe in the other highland areas of New Guinea (see table 4). This observation in subjects whose physical characteristics are similar suggests that CD^ue may have arisen in the area from the CDe chromosome rather than have been introduced by a migrant group. As *d* has not been found in New Guinea, it is likely that the D^u gene arose by mutation from D towards *d* rather than in the reverse direction.

Homozygotes for the mutant gene D^u are so far known among four different language groups of the Kainantu area, the Auiana, Tairora, Agarabi and Gadsup peoples, which suggests that the mutation occurred many generations ago. (See map, figure 1, and table 7.). These four

TABLE 7

Frequency of D^u homozygotes according to language group (groups in roughly west-to-east order)

	TOTAL SAMPLE	NO. HOMOZYGOTES	FREQUENCY
Gimi	25	0	0.0
Usurufa	38 + 23 = 61 *	0	0.0
Auiana	45	1	2.02%
Oiana (Ontenu)	64	0	0.0
Tairora	70	2	2.86%
Agarabi	54	2	3.7%
Gadsup	56	5 **	8.93% **
Total	375	10 **	2.93%

* Two Usurufa villages were sampled.

** One additional homozygote omitted as it did not occur in a random sample.

languages, although distinct, show unmistakable, if differing degrees of relationship to each other. Each language is spoken in a number of districts or villages. The blood samples for each of the four linguistically defined peoples came largely from the residents of a single village. Though not all of the individuals of any given village (in this area) are necessarily native to the village (the women, especially, may not be), all

of the 11 individuals showing the mutant gene D^u in these series are themselves native to their respective villages, as also are their parents. Thus there are no obvious or recent migrations to account for the present distribution of the mutant in the series.

It is not intended to suggest that contacts, including interbreeding, do not normally occur across linguistic boundaries in this part of the world. Quite the opposite may be true in the case of immediately adjacent groups or villages of different speech. (Data are as yet insufficient, however, to show whether language differences may constitute or contribute to a *relative* barrier among the peoples in question.) But while language *per se* is not an obvious barrier, there has been in the recent past and for as long as can be remembered a definite limit upon the radius of contacts, marital and otherwise, of any given village. The radius may be assumed to vary with conditions of terrain and local polity, but a figure of five to ten miles is probably a generous estimate for the average.

Between the Auiana village sampled and the Gadsup village sampled lies a distance of some 40 miles, aproximately, by existing footpaths. Contacts in the pre-European social and political world of Kainantu did not commonly—if ever—occur at such distances. The probability is thus lessened that simple intermarriage over a large area in a short span of time will explain the present distribution of mutant gene D^u among the groups sampled. It is safe to say, for example, that neither the Gadsup nor the Agarabi group had had, until recently, any contact whatsoever with the Auiana group nor probably any knowledge of this particular Auiana village. (In fact, the existence of the Auiana tongue with its perceptible similiarities to their own was quite a surprise to Agarabi when it first became known to them after European penetration of the area.) The possibility of intermediaries and intergrades between and among the four villages with the D^u mutant, resulting in a slow gene flow in space-time, is of course not ruled out.

To simplify the further discussion of the possible origin of the mutant, two hypothetical models are diagrammed below. The first we will call the "Linguistic Model" (figure 3). It will represent the present distribution of a mutant if the mutant first arose within a given speech community where, then and subsequently, mating takes place, with a frequency better than chance, among fellow speakers, though sometimes perhaps between mates whose birthplaces are at a considerable distance from each other. The mutant would then have been transmitted principally to descendants of the same community. The preponderant

majority of the present homozygotes of a mutant, such as D^u, would thus be speakers of the same or of cognate languages, the latter if sufficient time had elapsed for such languages to split off from the orginal speech community. Not all the cognate languages of the family, even so, would always or necessarily be represented among the homozygotes, of course; but the languages represented would tend to be more closely related to each other in time than any of them was to a cognate language whose speakers included no homozygotes for the mutant.

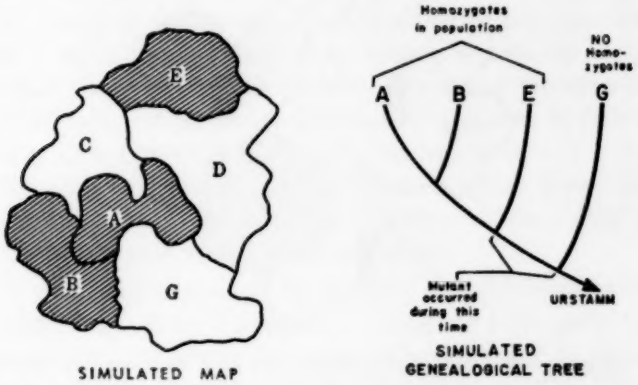

FIG. 3. LINGUISTIC MODEL OF THE DISTRIBUTION OF A MUTANT: when mating is primarily with speakers of one's own language. Hachure indicates presence of homozygotes in the population. Languages A, B, E, and G are related languages. However, the "gap" of time-depth separating A and G is greater than that separating any other pair of languages in the cognate group.

The inference from figure 3 is that the mutant arose subsequent to the splitting off of Language G from the *Urstamm*, or original parent tongue. Any dating of this event would thus place an outside limit on the age of the mutation. In this model, the spatial distribution of homozygotes other than in relation to language areas, is not primarily relevant for conclusions as to time.

The second model we will call the "Spatial Model" (figure 4). It will represent the present distribution of a mutant if the mutant first arose in a given area where, then and subsequently, mating takes place, with a frequency greater than chance, among persons living within a certain radius of each other, but without reference to barriers other than geographical ones or distance. The mutant would thus have been transmitted principally to persons of the same district, and if the district

happened to contain more than one language, cognate or not, the boundaries of these speech communities would not register appreciably on a gene distribution map.

The inference from figure 4, which diagrams a Spatial Model, is that the mutant first arose at a point near the center of its present distribution, which happens to be where the three language areas converge—unless geographical barriers suggest otherwise. The degrees of relationship and

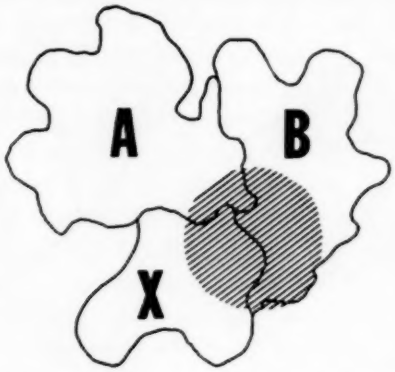

FIG. 4. SPATIAL MODEL OF THE DISTRIBUTION OF A MUTANT: when propinquity primarily determines mating. Hachure indicates presence of homozygotes in the population. Languages A and B, *e. g.*, are related. Language X is not related to either of them.

any dating of linguistic events in the area may tell nothing about the putative date of the origin of the mutant. The dispersion of the mutant is the principal index to its age in this model.

A third kind of situation is conceivable. If the mutant arises near the center of a large area occupied by a single language, and if its spread does not cross the boundaries of the language at any point, then, whether the mating principles tend toward the linguistic rule or toward the rule of proximity, all one can conclude is that the origin of the mutant is at most not older than the origin of the given language. (Even this conclusion, of course, excludes the possibility that a population now no longer distinct in, but earlier occupying, the area may have possessed the mutant in its gene pool and have passed on the mutant to its areal successors.)

The last possibility above is ruled out in connection with the present data on D^u in the Kainantu area of New Guinea, but both the Spatial

Model and the Linguistic Model may have some relevance. There are, however, at least two preliminary points to be made: (1) the two ideal models sketched above both assume a complete knowledge of the distribution of the mutant in question, as well as a knowledge of the extent to which mating according to language or mating according to propinquity is practiced. We do not at present have a complete knowledge of the distribution of D^u for the area in question and our knowledge concerning the rule of marriage is simply that propinquity plays a large part in it, although language may not be entirely ruled out as a factor. (2) Secondly, the ideal models diagrammed above do not take into account any differences in the frequency of the mutant as between one population and another among whom it is present. We do, on the other hand, have some—admittedly tentative—indications of variations in frequency. On the basis of such knowledge as we have, incomplete though it is, and with the considerations in mind that have been outlined above, an attempt will therefore be made to carry the question of the origin of the D^u mutant as far as may be presently practicable.

In assessing the degrees of relationship of the languages involved in the present blood series, reference will be made principally to the type of investigation now known as glotto-chronology and to certain recent glotto-chronological researches in the Kainantu area by Dr. Wurm of the Australian National University. The principal assumption upon which glotto-chronological calculations are founded is that a specified, "basic" part of the total vocabulary of a language changes at a constant rate over time. (Lees, 1953) By counting the number of cognates remaining in the basic list for two or more related languages, the period of time elapsed since the separation of the languages can thus be calculated in years. Table 5 presents some of Dr. Wurm's calculations. Even with a large margin of error, of course, the procedure gives a satisfactory estimate of relative degrees of linguistic relationship. Thus there is little question on any score that Gadsup and Agarabi are closely related languages, far more closely related than either is to Tairora; likewise Usurufa and Auiana are very close, especially when either of them is compared to Tairora. In broadest terms, glotto-chronological estimates reflect the net, cumulative response of two or more related languages to all the opportunities the languages have had either to differentiate from each other or to remain the same. Since this is essentially the same kind of question that we are pondering on the genetic level, there may be

some point in discussing the degrees of linguistic relationship among the populations indicated.

A criticism sometimes leveled at the theory of glotto-chronology is that the various opportunities for languages to diverge (or to remain the same) may not, in fact, produce a constant rate of change. For example there could conceivably be secondary reborrowing between two languages long separated in space and subsequently relocated in close proximity to each other. Tairora might serve for illustration of this if it has had substantial recent borrowing from Agarabi or Gadsup, its once close relatives and recent neighbors. In such a case the number of cognates in the basic vocabularies of the two languages could lead to an underestimate of the actual length of separation of the languages. From our point of view such a possibility would certainly not constitute a basic flaw in the method, however, since we are interested in assessing contacts, and hence opportunities for gene flow, whether the contacts were aboriginal, early, recent, or current.

It may be noted that homozygotes for D^u do not occur in a Gimi series, albeit the series is a small one of only 25 individuals. The Gimi language is only remotely related to the languages of the peoples so far showing the mutation. The Gimi area is also a considerable distance to the west of the others sampled and is separated from them by intervening peoples. The negative findings from Gimi (and, of course, at greater remove, the negative findings from nearly all previous New Guinea Highland blood series to the west of the Kainantu area, see table 4) would be consistent with an historical-genealogical locus of the mutation in some way associated with linguistic affiliation as well as with geography. In a sense our general problem is the extent to which these two dimensions have not been functionally equivalent.

Perhaps the most salient problem in the present blood series, as seen from the point of view of the languages represented, is the absence of homozygotes for D^u in the relatively large Usurufa series and in the Ontenu (Oiana) series. These populations speak languages closely related to those of Auiana-Agarabi-Gadsup, three populations having D^u homozygotes. At the same time, D^u homozygotes were present among the Tairora, who are much less immediately related to the others. The negative findings from Usurufa and Ontenu (Oiana), as well as the positive one from Tairora, both indicate again that the linguistic dimension is not the only one and that there is a distinct spatial or geographical dimension. Specifically these findings suggest that D^u mutants initially

occurred to the east of the Usurufa area. Moreover, on this and other grounds, they probably did not occur first among the Auiana. Conceivably the mutants might have occurred first among the Tairora or some other people outside the present series with whom the Tairora are or have been in contact. The Tairora hypothesis would be tenable, presumably, only if it were found that the Gadsup had also been in contact with the same outside people(s). One is tempted to rule out the Kamano for the origin of the mutant, even without a Kamano blood sample. The grounds for this are much the same as for ruling out the Auiana: if the mutant had originated among them and were generally present in their population, the absence of homozygotes among—despite intermarriage with—the neighboring Usurufa and Ontenu (Oiana) would be hard to explain. The only obvious exception would be if the contacts of Auiana or Kamano are more recent with Usurufa and Ontenu (Oiana) than with any of the other peoples where D^u homozygotes are present.

By far the highest frequency of homozygous D^u individuals among the present series occurs in the Gadsup sample, the easternmost group. The absence of homozygotes in the Ontenu (Oiana) sample is therefore somewhat puzzling because these people are not only closely related in language to the Gadsup but themselves consider the difference to be slight. (The Ontenu village from which the sample came are in constant contact and exchange women in marriage with some Gadsup villages, although probably not very often with the Arona village or district from which our particular Gadsup blood samples come.) But excepting the Ontenu (Oiana) group, there is otherwise a steady west-to-east increase from Auiana to Gadsup in the percentage of D^u homozygotes. (An even more consistent east-to-west cline was noted above for positive reactors to anti-S, table 6.) Granted the samples are small and scattered, the total populations and the area large, the consistency of the west-to-east trend is nonetheless suggestive. The frequency of homozygotes in the Gadsup sample is over twice the nearest figure, that for their neighbors and close linguistic relatives to the west, the Agarabi; and the Gadsup frequency is over four times the frequency of the Auiana, the westernmost sample possessing D^u in the series. The Tairora are about midway between Agarabi and Auiana in frequency of D^u homozygotes. In sum, a spatial dimension is suggested by the present data on D^u, as well as S, one having an east-west gradient.

At the same time, the languages represented among the known D^u homozygotes are all within what Wurm (unpublished Ms., 1959) calls

the Gadsup-Usurufa-Tairora Family. Within the Family, Wurm recognizes three sub-families, Gadsup, Oiana, and Agarabi belonging to one; Usurufa and Auiana to the second; and Tairora to the third one mentioned—by far the most distantly related of the three. (Wurm's estimates of the time-depth separating some of these languages are given in table 5.) If further blood sampling in the Kainantu area confirms the primary historical association of D^u with the Gadsup-Usurufa-Tairora Family of languages, or with certain portions of it, it may be possible to use the figures given for language time-depths to bear upon the question of the time-depth of the D^u mutation. Were it true that we confront here a situation approximating the Linguistic Model, we could seriously consider the figure of 2350-2750 years which separates Tairora from Auiana, the largest "gap" or time-depth among the languages represented by known D^u homozygotes. Since present data suggest that our situation is more complex than the Linguistic Model, however, the need for fuller information is all the more important before an answer to this question can be carried further.

SUMMARY

The ABO, MNS and Rh blood groups were determined on 375 native subjects from Kainantu, New Guinea, and the gene frequencies were calculated. The general pattern is similar to that found in other parts of the New Guinea Highlands, except for the genotype CD^ue/CD^ue in eleven of the subjects. The gene D^u is presumed to be a mutation from D towards *d*. Its origin and distribution in the Kainantu area is discussed. Three of four CD^ue/CD^ue subjects who were tested failed to react with anti-C^N serum, so that the gene C^n must be present at the C locus in these subjects. The C^n gene was also found in five out of 38 subjects from another part of New Guinea, all of whom reacted with anti-C and anti-D.

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DURATION OF ILLNESS *

BY H. M. C. LUYKX AND BETTY L. MURRAY

THE duration of an illness is one of its significant epidemiological characteristics. It gives information about a particular case which is fully as important as its place of occurrence, the season of the year, or the age or sex of the patient, for example. Duration is a most convenient practical measure of severity of a disease. In this study this is particularly true, because two of the more obvious measures of severity, fatal or non-fatal termination, and the presence or absence of complications, have been eliminated by selecting only uncomplicated cases returned to military duty. Duration is also a fairly reliable measure of the cost of an illness. It describes the loss of earning power of the patient and the economic loss to the community quite accurately, and it correlates highly with the out of pocket expense to the patient, or, in the case of military personnel, to the government.

For a given disease, then, its mean duration is a useful piece of information; and for a large number of cases, drawn from a homogeneous population, it can be stated with a high degree of reliability. But the features of military medical records which contribute to the reliability of mean durations also provide a unique opportunity to do more than observe this single parameter. The frequencies are greater, the source population is more homogeneous, and the accumulated records are more uniform, than is probably true of any other body of clinical data. Hence other descriptors of duration distributions are worth observing, and the body of epidemiologic information for specific diseases can be significantly increased thereby.

In plotting distributions of durations for any definable group of cases it is immediately apparent that they are skewed, sometimes more, sometimes less, but always to the right. Thus a single centering constant and a single measure of spread do not describe the data adequately. Let us therefore consider a few of the ways in which duration distributions

* From the Biometrics Division, Office of the Surgeon General, Headquarters U. S. Air Force. This paper contains personal views of the authors and is not to be construed as a statement of official Air Force policy.

can be presented. A simple histogram or frequency polygon on a scale of duration would be useful. It would show the mode and the skewness at a glance. But the choice of class intervals would affect the clarity of the picture (see figure 1), and the median and other percentile

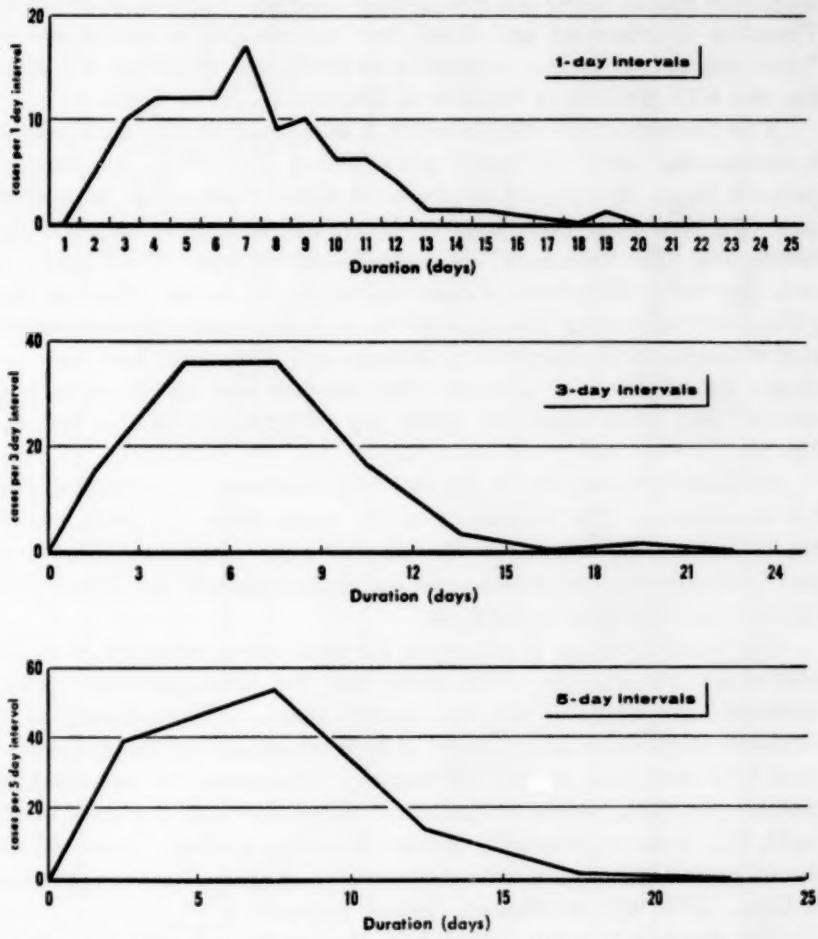

FIG. 1. MEASLES (RUBEOLA) WITHOUT PNEUMONIA, 107 SINGLE DIAGNOSIS CASES RETURNED TO DUTY, U. S. AIR FORCE MILITARY PERSONNEL: 1954-56. (Three distributions of the same data.)

points could not be read directly from the graph. The solution to these difficulties is to plot cumulative frequencies. Ordinates would then measure the number of cases discharged "to date" (in days after

admission to medical care). Converting the frequencies to the percent discharged to date would obviously be even more useful, so that each curve would approach the 100% mark asymptotically. However, a physician or hospital administrator is more interested in knowing how long cases will be under his care or how long his beds will be occupied. Therefore distributions are shown here as negative accumulations or "cases remaining" curves, beginning at 100% and dropping to 0 when the case with the longest duration is disposed of. (See figure 2.)

This duration curve (for measles) is seen to be strictly analogous to a survivorship curve commonly given with a life table. A cohort of patients begins its episodes of illness at time of admission to medical care (the zero point on a scale of days), and diminishes as each case terminates. The "staircase" is drawn to aid the eye. The "riser" of each step shows the percent of cases discharged during the following day. (The curve connecting the edges of the steps represents the assumption that patients are discharged at a uniform rate throughout each day, not always literally true of course.) The steepest part of the curve (the tallest "riser") indicates the modal day of discharge, in this case the 7th day.

Ordinate values of 75, 50, 25, and 10% are drawn as horizontal lines for convenience. The median is at the point where the curve crosses the 50% line, shown here as the 7th day after admission. The 25th and 10th percentile values for cases remaining similarly are shown to be the 9th and 11th days respectively.

The mean duration is computed for each curve, although it is not plotted on the abscissa. This mean and the three percentile points mentioned are shown in the box on each chart. (There should be no confusion in speaking alternatively of 25% remaining or 75% discharged, or of 10% remaining or 90% discharged.) Comparing the mean and the median provides a notion of skewness. Since skewness is always to the right, the mean is invariably greater than the median. Some of the duration distributions are nearly symmetrical, but in others the skewness is high. This will be discused further presently.

The number of cases included in the graph is also shown, which gives a notion of the reliability of the curve, and which is the basis for the 2σ confidence intervals given. Two standard errors above and below the mean are computed in the usual fashion. For the 107 measles cases these give a range from 6.3 to 7.5 days around the mean of 6.9 days. The computation of the interval from 2σ below to 2σ above any percentile point is described in the appendix to this paper. Basically it amounts

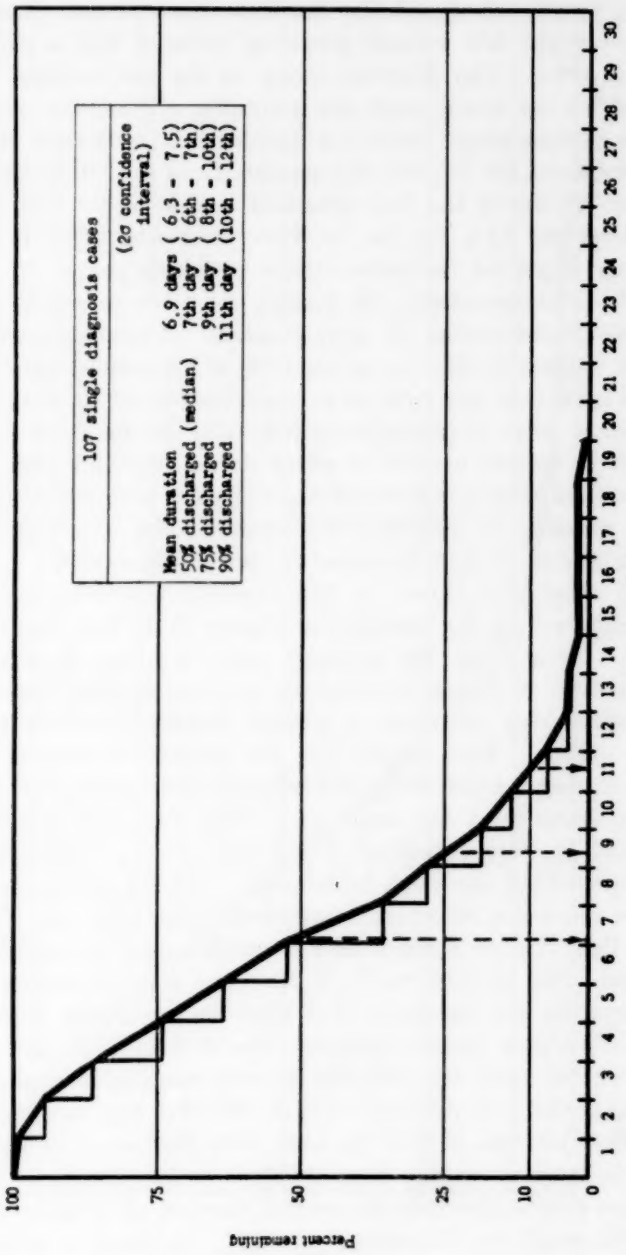
107 single diagnosis cases
(2σ confidence interval)
Mean duration 6.9 days (6.3 - 7.5)
50% discharged (median) 7th day (6th - 7th)
75% discharged 9th day (8th - 10th)
90% discharged 11th day (10th - 12th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
Days after admission

FIG. 2. MEASLES (RUBEOLA) WITHOUT PNEUMONIA, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

to calculating how far above or below this curve (in a vertical direction) another curve might fall without providing evidence that a different universe is involved. The intervals shown in the box indicate where these 2σ-limit curves would reach the percentile ordinate in question. Thus for the measles graph, there is a confidence interval from the 6th to the 7th day about the 7th day as a median, from the 8th to the 10th day about the 9th day as the 75th percentile, and from the 10th to the 12th day about the 11th day as the 90th percentile. The intervals usually become larger for the more extreme percentile points.

For the hospital commander, the median and other percentile points are more useful statistics than the mean duration. It means more to him to know that it takes 11 days to return 90% of his measles patients to duty than to know that they have an average duration of 6.9 days. The percentile points (with their confidence intervals) are shown in the box on the graph in ordinal number of whole days. Although computing technique provides these points in fractions of days, such fractions have no practical meaning in hospital administration, and rounding to the nearest whole day would be bothersome for the administrator.

Figure 3 shows the curve for 368 cases of infectious hepatitis. This graph differs from the measles one (figure 2) in only one important respect. Because of the normally long duration of infectious hepatitis, the scale of days after admission is given in 5-day instead of 1-day intervals. This introduces a slightly different meaning to the "staircase" plotted. Each "riser" is the percent discharged from medical care during the next 5-day interval, and the steepest part of the curve thus indicates the 5-day modal class. The computed points however are based on frequencies for 1-day intervals, so that they are strictly analogous from one graph to the next. (All the graphs prepared in this series are scaled either at 1-day or at 5-day intervals. Visual comparisons therefore are made as easy as possible, and the reader need only—he must—first observe which of these two scales is used.) For infectious hepatitis for the years 1954-1956 the mean was 45.8 days with the 2σ confidence interval ranging from 42.8 to 48.7 days. The median was on the 42nd day with the interval ranging from the 39th to the 46th day; the 75th percentile was on the 58th day, ranging from the 55th to the 61st; and 90% of the cases were discharged by the 75th day, with a 2σ interval ranging from the 72nd to the 81st day.

A few more graphs illustrate the general shape of all of these curves. Figure 4 is the graph for 446 cases of mumps. The mode is at the 7th or 8th day, the median at the 10th day, and the mean is computed as

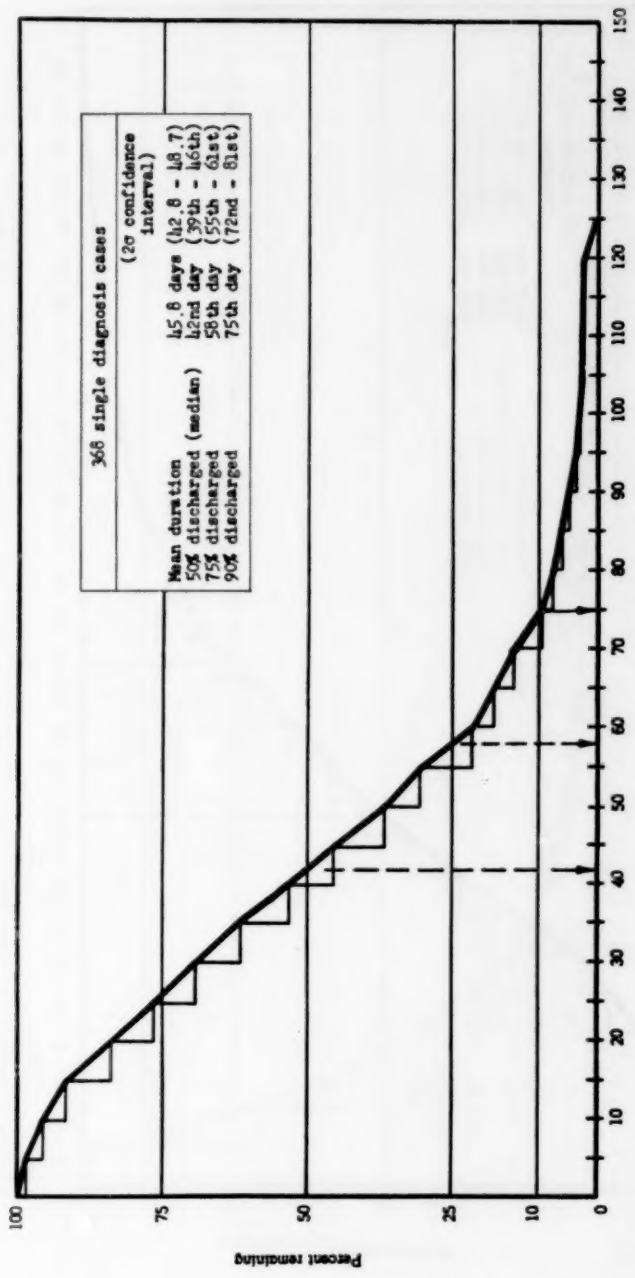

FIG. 3. INFECTIOUS HEPATITIS, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

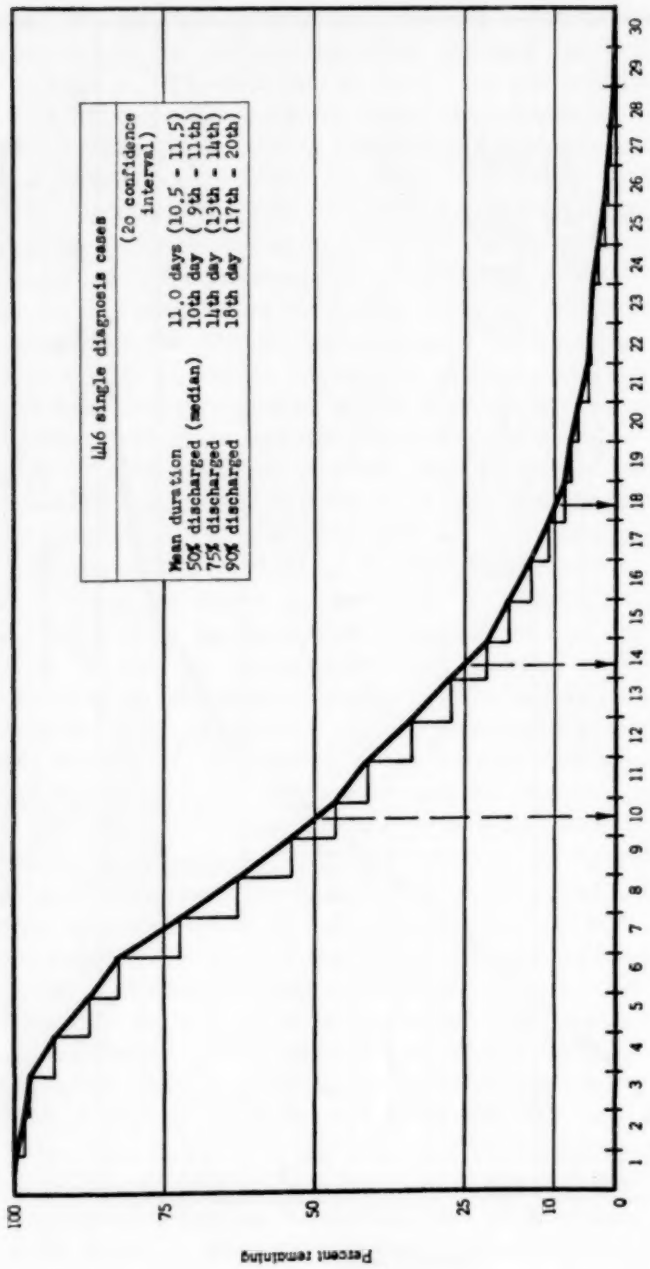
446 single diagnosis cases
(20 confidence interval)
Mean duration 11.0 days (10.5 - 11.5)
50% discharged (median) 10th day (9th - 11th)
75% discharged 14th day (13th - 14th)
90% discharged 18th day (17th - 20th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
Days after admission

FIG. 4. MUMPS, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

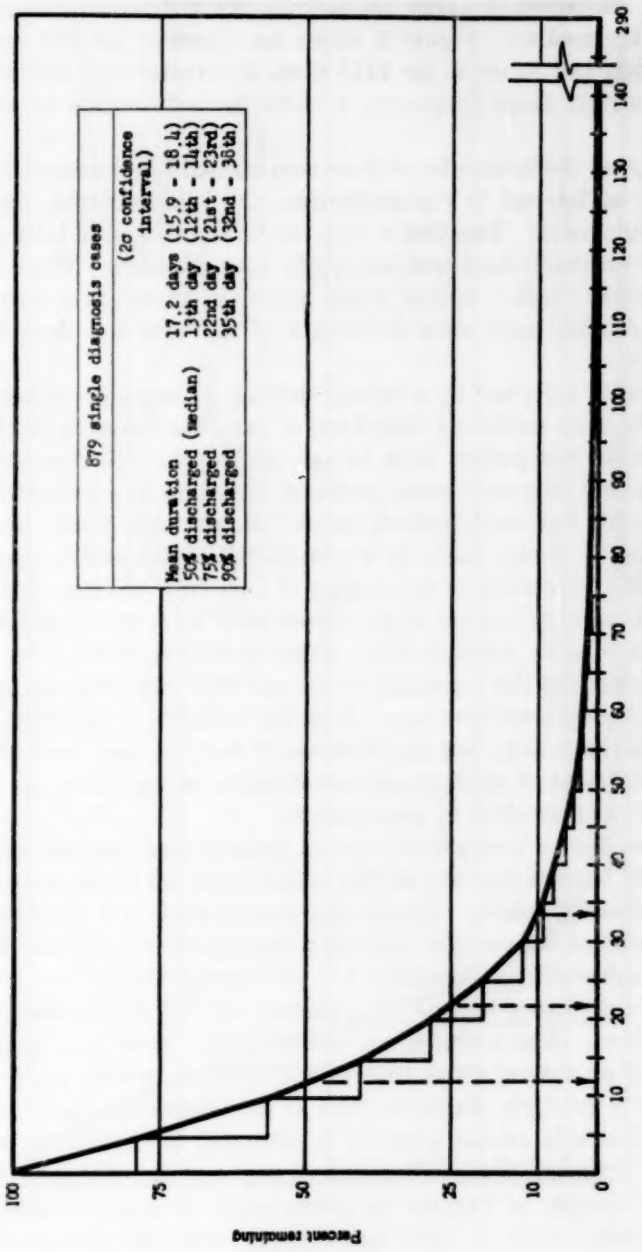

FIG. 5. PILONIDAL CYST, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

11.0 days. Skewness is again revealed by the difference between the mean and the median. Figure 5 shows the duration for 879 cases of pilonidal cyst, and figure 6, for 2177 cases of streptococcal sore throat. With a relatively large frequency, a curve naturally tends to become smoother.

Two general characteristics of these curves should be discussed briefly, as they are of interest to the statistician, the epidemiologist, and the hospital commander. The first is that there are always at least a few cases of surprisingly short and unusually long duration. What is the reason for these "tails," as they would appear in an ordinary frequency polygon? At this point some definitions of the data and their source are necessary.

Each case is described by a record showing, among other things, the diagnosis, with its statistical classification code, the duration of illness, and facts about the patient such as age, and so on. The record is a standard clinical summary form, prepared according to a prescribed set of instructions, for every patient treated at any Air Force hospital. The diagnosis is always made by a qualified physician and is therefore highly reliable. Duration is the number of days from the time a patient is excused from duty because of his illness until he is discharged by his doctor for return to military duty. This definition allows very little leeway in specifying the beginning or the end of a case of illness, and is therefore a highly consistent one. It is the duration of disability with respect to military duty, not the duration of hospital stay, even though in the management of military patients duration of disability and duration in hospital may often by synonymous.

In the interest of homogeneity certain types of cases are not included in this study, because they are readily identified as not being comparable with the bulk of the cases. Deaths and persons separated from the Air Force for medical reasons are excluded; they constitute only about 2% of all patients. Their durations are not comparable with those of returned-to-duty cases, because the illnesses are not terminated in the sense used here. Also excluded are records which show more than one diagnosis for an illness, about 10% of the returned-to-duty cases. The duration of a multiple diagnosis case is the length of illness of the patient, and usually cannot properly be allocated to one of the several diagnoses. Although the combination of two specific diagnoses might occur often enough to warrant its presentation as a single diagnostic entity, this can rarely be done conveniently with the present record processing system.

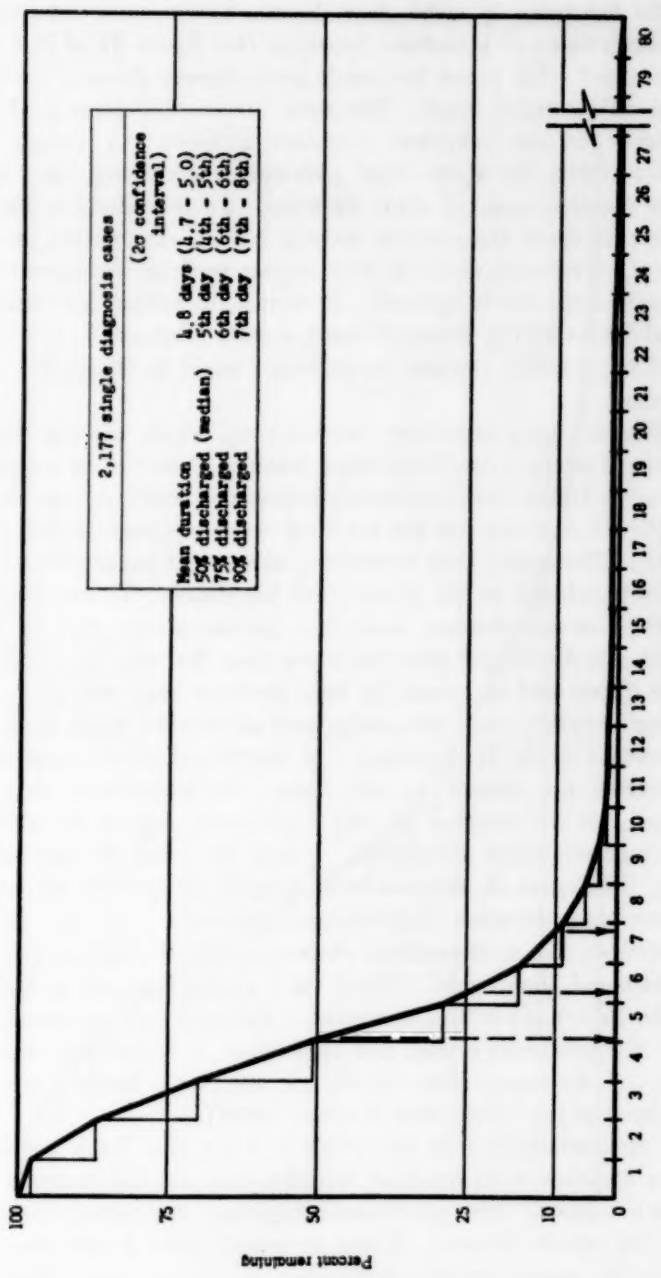
2,177 single diagnosis cases
(2σ confidence interval)
Mean duration 4.8 days (4.7 - 5.0)
50% discharged (median) 5th day (4th - 5th)
75% discharged 6th day (6th - 6th)
90% discharged 7th day (7th - 8th)
Percent remaining
100
75
50
25
10
0
Days after admission
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 79 80

FIG. 6. STREPTOCOCCAL SORE THROAT, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

With the foregoing in mind, how do we explain such observations as a half dozen cases of infectious hepatitis (see figure 3) of less than 5 days' duration? The graph for nearly every disease shows a few cases which seem unreasonably short. For some diseases the records of such cases probably do not represent complete illnesses. A person may unexpectedly return for a few days' post-convalescent care, thus being recorded as a second case, of short duration. In this study it has not been possible to check the original records for an explanation, so that the chance of an erroneous record, with respect to either duration or the diagnosis code, must not be ignored. However, the system for reviewing records and for verifying punched cards is well established, and errors alone could not possibly explain the extremes noted in practically every duration curve.

The unusually long cases may include some which are complicated by a concurrent medical condition which was not identified by a separate diagnosis code. In the record reviewing system this could perhaps slip by. More significant, however, are the rules for recording convalescent leave. If a patient is discharged from treatment, subsequent convalescent leave, if any, is not included in the duration of his illness. If, on the other hand, he receives convalescent leave in a patient status, that is, he is re-examined and discharged after his leave, then the leave is included as part of the illness and an unusually long duration may result.

The long duration cases are really part of another point to be discussed: skewness of the distributions. As mentioned before, most of the duration curves are skewed to the right. It is doubtful that this phenomenon can be ascribed in any significant respect to artifacts resulting from reporting procedures. From the point of view of the statistician, the degree of skewness is of interest along with other parameters describing duration distributions generally. At the present writing, however, the mathematical characteristics of these curves have not been explored thoroughly. Third- and higher-moment coefficients, for example, have not yet been computed. Primarily we are developing a tool for the physician's use, and if needed, a sufficiently accurate description of skewness here would be the well known formula: $3(\text{mean} - \text{median})/\sigma$ (Yule and Kendall, 1940).

To the epidemiologist it is important to know that there are nearly always long duration cases reported, but that they are less common than short duration cases. For all diseases together, for broad diagnostic classes, or for specific diseases, it has invariably been found that cases shorter than the mean duration outnumber the longer ones. The three

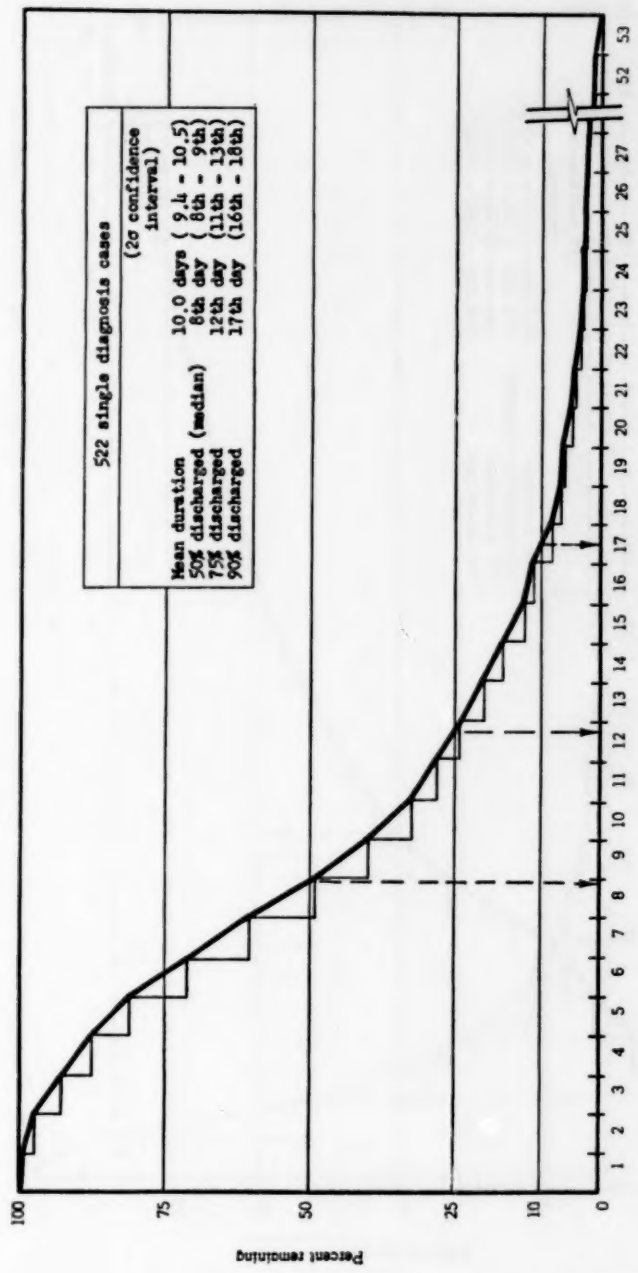
522 single diagnosis cases
(2σ confidence interval)
Mean duration 10.0 days (9.4 - 10.5)
50% discharged (median) 8th day (8th - 9th)
75% discharged 12th day (11th - 13th)
90% discharged 17th day (16th - 18th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 52 53
Days after admission

FIG. 7. BRONCHOPNEUMONIA, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

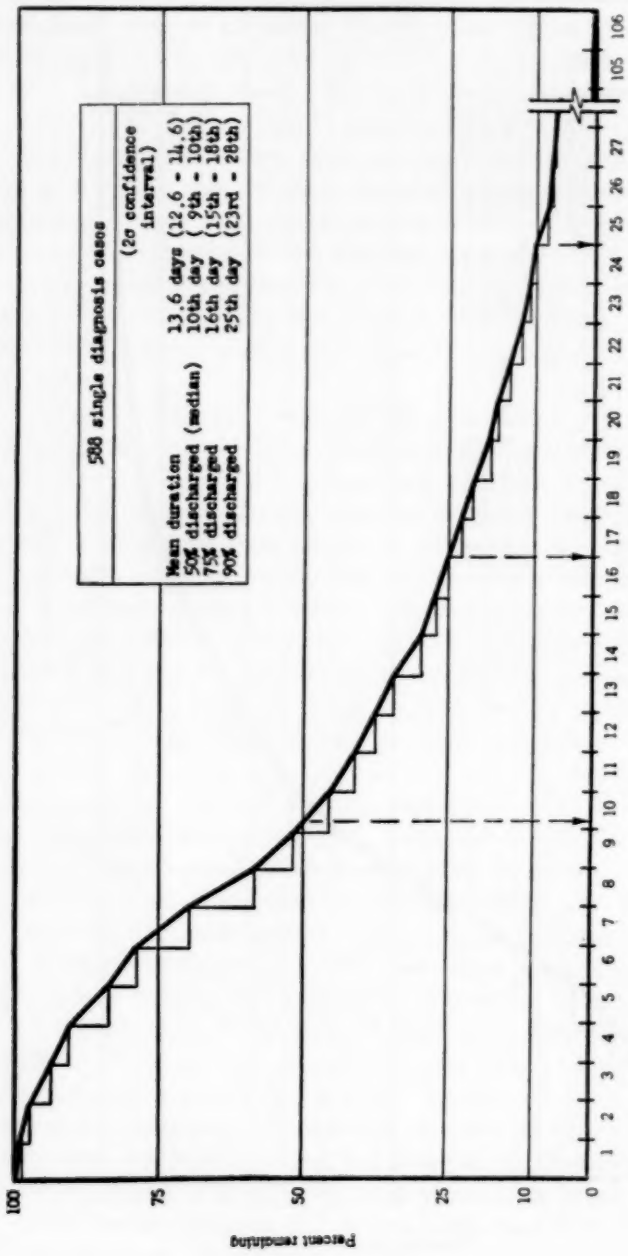
588 single diagnosis cases
(2σ confidence interval)
Mean duration 13.6 days (12.6 - 14.6)
50% discharged (median) 10th day (9th - 10th)
75% discharged 16th day (15th - 18th)
90% discharged 25th day (23rd - 28th)
Percent remaining
100
75
50
25
10
0
Days after admission
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 105 106

FIG. 8. PRIMARY ATYPICAL PNEUMONIA, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

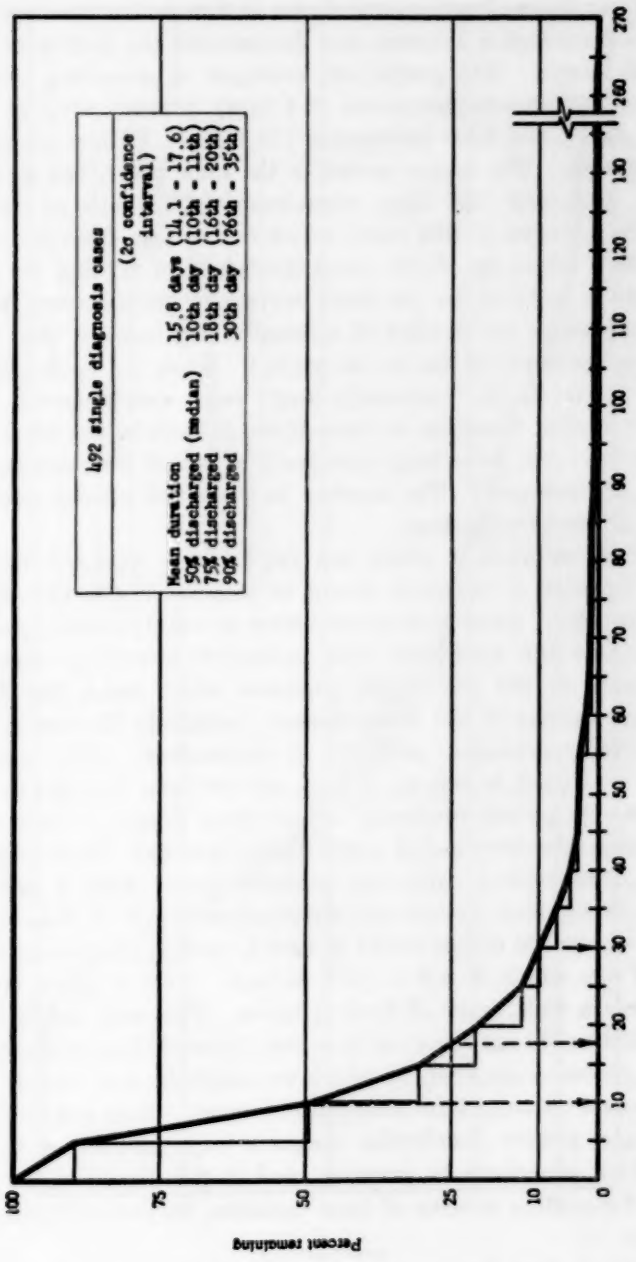

FIG. 9. LOBAR PNEUMONIA, AIR FORCE MILITARY PERSONNEL RETURNED TO DUTY: 1954-56.

curves for three types of pneumonia shown in figures 7-9 illustrate some points of epidemiological interest, and demonstrate the particular value of durations curves. The graphs are arranged in ascending order of median duration: bronchopneumonia (8.4 days), primary atypical pneumonia (9.7 days), and lobar pneumonia (10.4 days). There are around 500 cases of each. The means ascend in the same order, but in bigger steps: 10.0, 13.6, and 15.8 days, respectively. This tells us that the skewness also increases in this order, which can be seen from the shapes of the curves. (Here the slight visual gymnastic of reading the 5-day interval scale is required for the third curve. It was necessary to plot on that scale because the number of unusually long cases of lobar pneumonia skews the curve so far to the right.) What the epidemiologist might now ask is: If the "unusually long" cases were removed, would the mean or median durations in these three diseases be the same? Or put another way: Are these long cases really a part of the universe with which they are grouped? The answers to these and similar questions are left to future investigation.

The writers entertain a belief, not yet properly explored, that the shape of a duration distribution should be associated with the state of medical knowledge. Often a diagnostic term is simply a name given to a group of signs and symptoms, with incomplete knowledge about the etiologic agents or the physiologic processes which cause the illness. Examples are various of the virus diseases, metabolic diseases such as gout, essential hypertension, arthritis, or rheumatism. (For many of these, cases as defined in this study have not yet been reported in sufficient numbers to permit preparing one of these graphs.) One might ask the question: Are they in fact single disease entities? Would separate curves for more refined categories produce curves with a narrower spread, or with significantly different duration patterns? A well-defined, thoroughly understood disease ought to have a narrower durations distribution than one which is not so well defined. Even a given type of fracture shows a wide range of healing times. This may simply be an indication that we do not know, or have not explored thoroughly, all the physiologic processes involved, by which we might further classify fractures or fracture patients into homogeneous types. More generally, for certain diseases a wide distribution means a large number of biologic variables which contribute to duration, and if the disease entity were sub-classified according to some of these variables, narrower distributions should result.

Figures 10-13 show four curves of particular interest to the surgeon

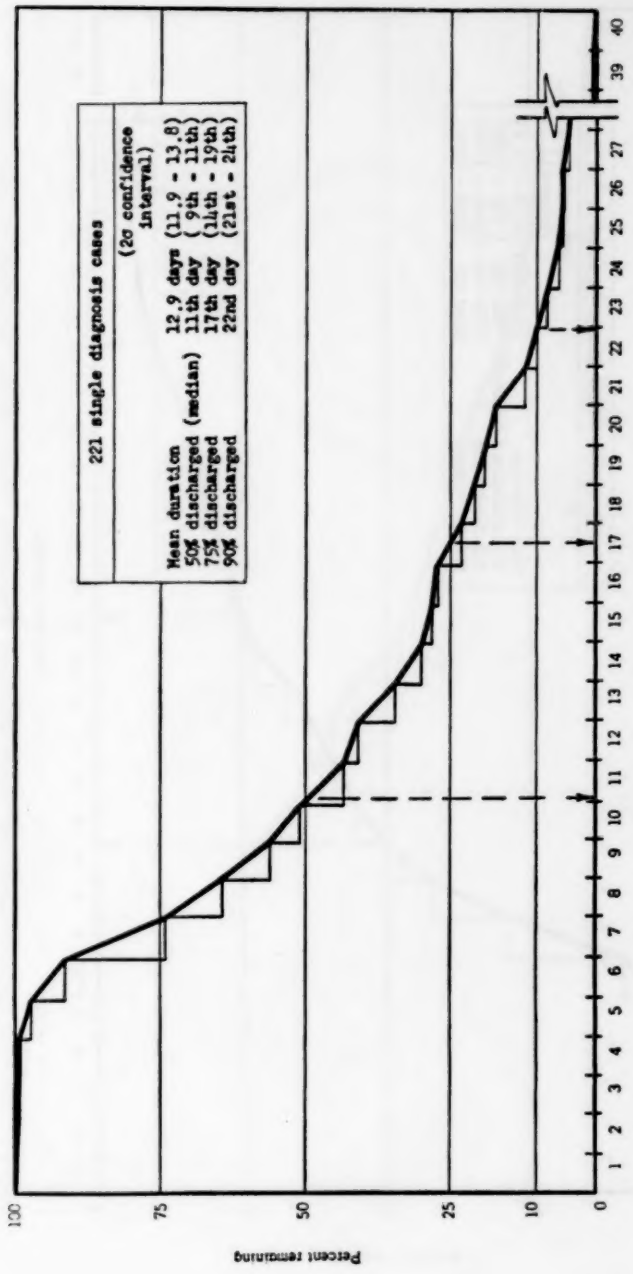
221 single diagnosis cases
(2σ confidence interval)
Mean duration 12.9 days (11.9 - 13.8)
50% discharged (median) 11th day (9th - 11th)
75% discharged 17th day (14th - 19th)
90% discharged 22nd day (21st - 24th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 39 40
Days after operation

FIG. 10. INGUINAL HERNIOPLASTY, AIR FORCE MILITARY PERSONNEL (AGES UNDER 20) RETURNED TO DUTY: 1958.

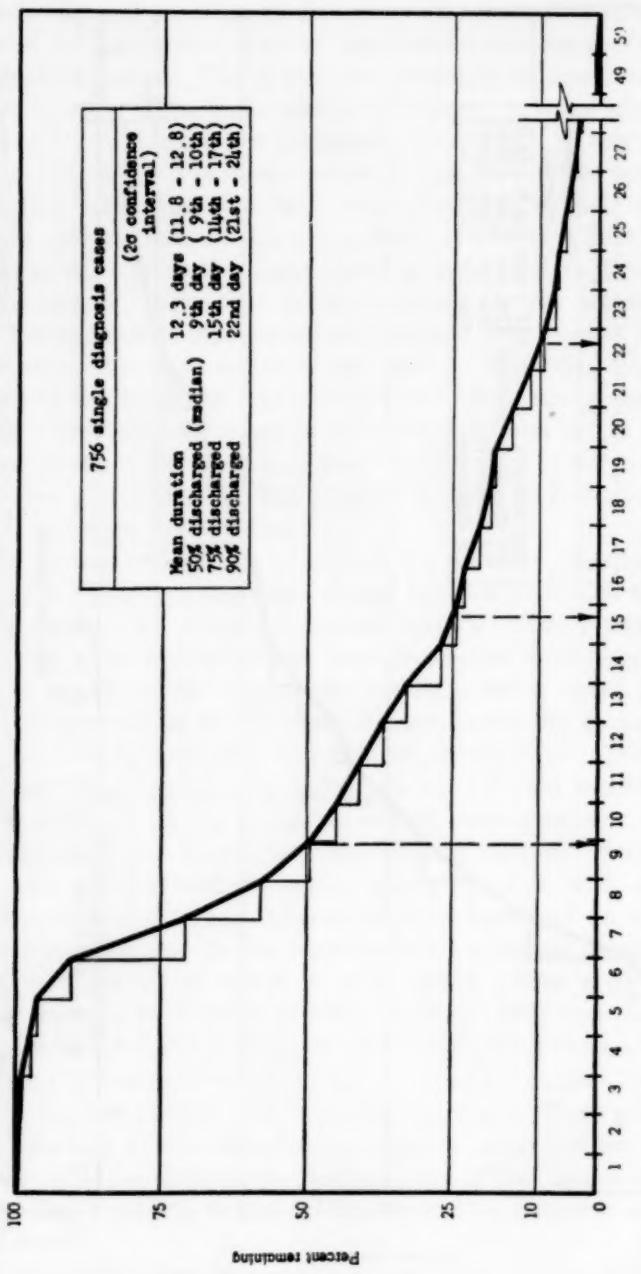
756 single diagnosis cases
(2σ confidence interval)
Mean duration 12.3 days (11.8 - 12.8)
50% discharged (median) 9th day (9th - 10th)
75% discharged 15th day (14th - 17th)
90% discharged 22nd day (21st - 24th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 49 50
Days after operation

FIG. 11. INGUINAL HERNIOPLASTY, AIR FORCE MILITARY PERSONNEL (AGES 20-24) RETURNED TO DUTY: 1958.

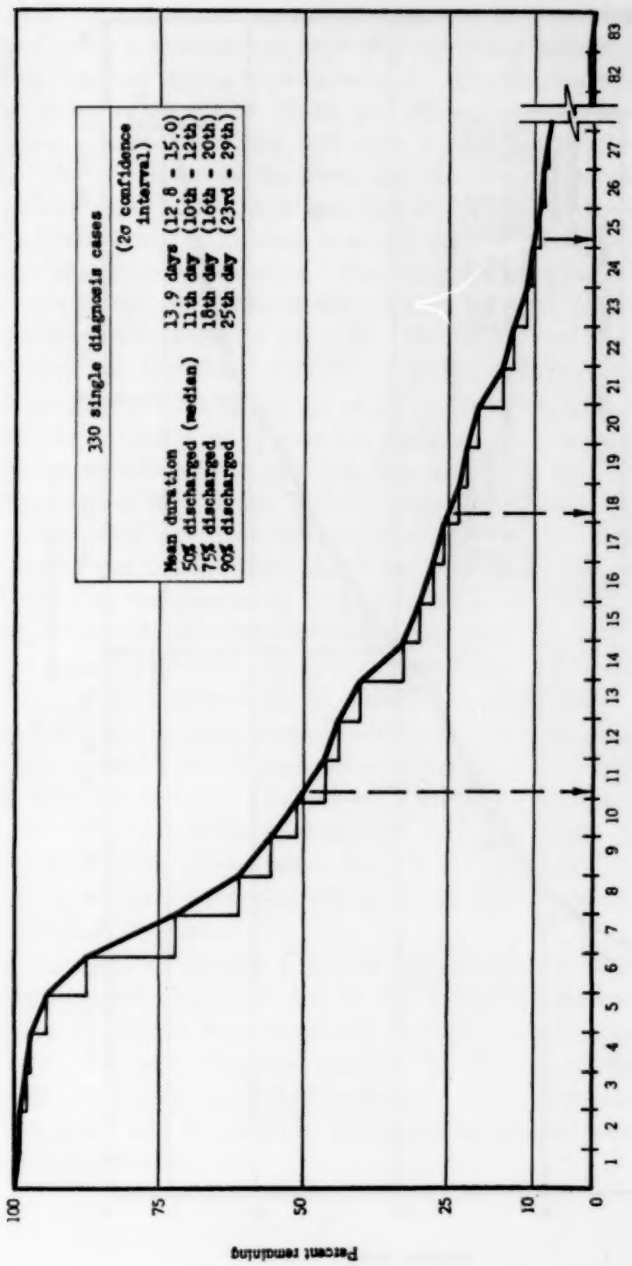
330 single diagnosis cases
(2σ confidence interval)
Mean duration 13.9 days (12.8 - 15.0)
50% discharged (median) 11th day (10th - 12th)
75% discharged 18th day (16th - 20th)
90% discharged 25th day (23rd - 29th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 82 83
Days after operation

FIG. 12. INGUINAL HERNIOPLASTY, AIR FORCE MILITARY PERSONNEL (AGES 25-29) RETURNED TO DUTY: 1958.

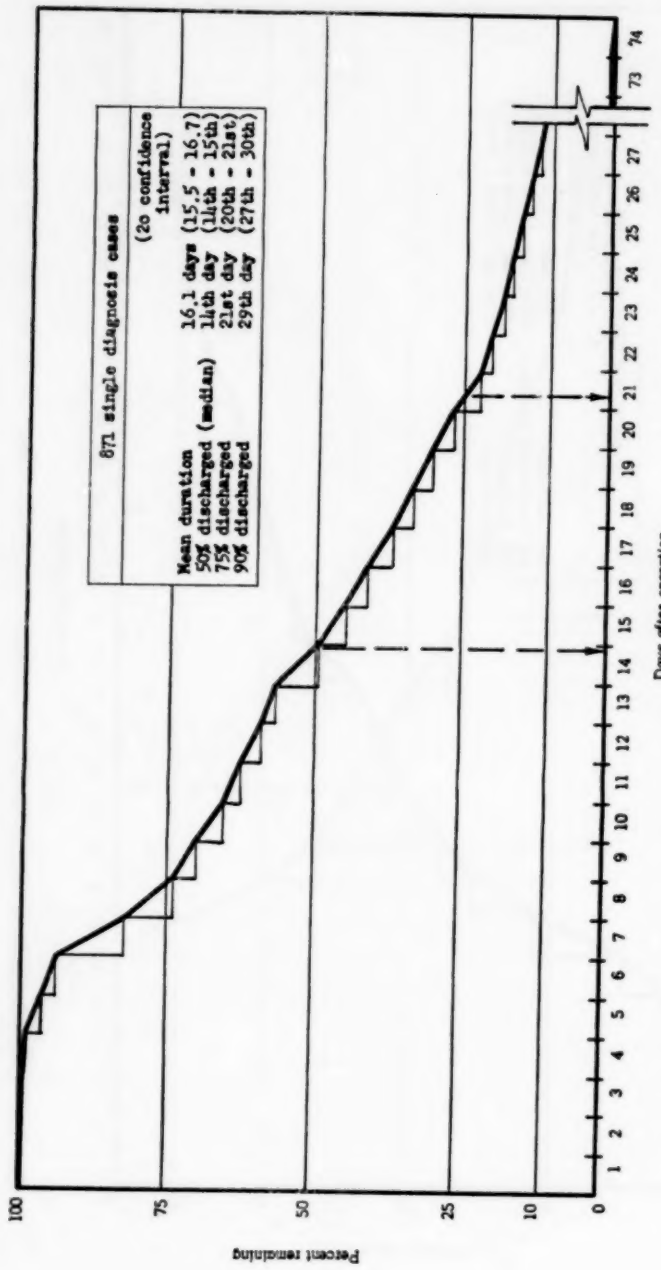
671 single diagnosis cases
(2σ confidence interval)
Mean duration 16.1 days (15.5 - 16.7)
50% discharged (median) 14th day (14th - 15th)
75% discharged 21st day (20th - 21st)
90% discharged 29th day (27th - 30th)
Percent remaining
100
75
50
25
10
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 73 74
Days after operation

FIG. 13. INGUINAL HERNIOPLASTY, AIR FORCE MILITARY PERSONNEL (AGES 30 AND OVER) RETURNED TO DUTY: 1958.

or hospital commander. Inguinal hernioplasty is a surgical operation, and the abscissa is measured in days after operation instead of days after admission, because that is what is wanted. The four curves are for the age groups under 20, 20-24, 25-29, and 30 and over. There were over 2000 cases, with no less than 220 cases in any one of these four age groups. The graph shows the mean and the median durations for the second group (ages 20-24) to be less than for the first group (under 20); a test for statistical significance, however, tells us this could easily have been due to sampling variation. The apparent association of average durations with age, therefore, is not inconsistent with the notion that it takes older persons longer to get well. The strength of the evidence in favor of such an hypothesis, however, requires further study.

For an ailment such as hernia, which is easily repaired, the military surgeon's responsibility is to return the patient to duty as soon as possible. His interest therefore is in the long duration cases, and he notes that in any age group 90% of his patients ought to go back to duty by the 24th to the 30th day (see the upper confidence limits of the 90th percentiles of the distributions). For his purpose, further investigation should look into the remaining 10%, perhaps to see whether they differ with respect to clinical characteristics other than age, such as weight, type of surgical procedure, or type of anesthetic. With large enough frequencies, these questions can be approached statistically.

The Air Force Surgeon General's Office is currently compiling duration distributions for each disease for which 100 uncomplicated, returned-to-duty cases are reported. At present a number of graphs such as the ones shown here have been drawn and are on hand. Some of these have been presented in an earlier paper (Luykx and Murray, 1958). As the years go by, larger frequencies will be accumulated and more diseases can be added to the list.

It is our hope to compile a sort of encyclopedia of duration curves. They can represent groups of cases in any desired categorization. For a specific disease, if the total frequency is large enough, separate curves for age, sex, and race categories would be of interest; or for particular geographic areas, or for method of treatment. And comparison of successive years also would be useful to the epidemiologist, to note a trend in the durations pattern.

APPENDIX

Computing Percentile Points, and the 2σ Confidence Limits for any Percentile Point, of a Frequency Distribution

The xth percentile point for a series of values is the scale value below which x percent of all the values lie. That is, the 25th percentile point is the value below which lie 25 percent of the values in a series; the 90th percentile point is the value exceeded by 10 percent of the given series of values. The 2σ confidence limits of a percentile point are those values between which there is reasonable assurance that the corresponding true percentile point lies. (There is less than 1 chance in 20 that the true point lies outside these limits.) Determining the confidence limits involves the computation of the standard error (σ) of the percentile frequency in qestion.

The standard error of a percentile frequency, for any type of frequency distribution, is obtained by the formula (Woodruff, 1952)

$$\sigma_{WN} = \sqrt{\sigma_1^2 + F^2\sigma_2^2 + 2F\sigma_{12}} \qquad (1)$$

where σ_{WN} = the standard error of the number of items in the true percentile frequency

σ_1 = the standard error of the number of items up to but not including the percentile class (the class interval containing the percentile point in question)

σ_2 = the standard error of the number of items in the percentile class

σ_{12} = the covariance between these two numbers

F = the proportion of the percentile class which is assumed to lie below the percentile point.

$$F = \frac{WN - C}{V}$$

where W = the fraction for which the percentile is named

N = the number of items in the sample (the total frequency) (WN is the percentile frequency)

C = the cumulative frequency up to but not including the percentile class, and

V = the frequency in the percentile class.

For computation, (1) is rewritten with the following substitutions:

$$\sigma_1^2 = NP_1Q_1 = C - C^2/N$$

$$\sigma_2^2 = NP_2Q_2 = V - V^2/N$$

$$\sigma_{12} = -NP_1P_2{}^* = -CV/N$$

since $P_1 = C/N$ = the proportion of items up to the percentile class

$Q_1 = 1 - C/N$

$P_2 = V/N$ = the proportion of items in the percentile class

$Q_2 = 1 - V/N.$

Thus
$$\sigma_{WN} = \sqrt{(WN - C)^2/V + C - W^2N} \qquad (2)$$

The 2σ confidence limits are determined by computing the scale values corresponding to the frequencies:

$$A = WN - 2\sigma_{WN} \qquad (3)$$

$$B = WN + 2\sigma_{WN} \qquad (4)$$

In algebraic terms, the interpolation for a percentile point in a frequency distribution is done by the formula

$$w = k + i(WN - C)/V \qquad (5)$$

where k = the lower limit of the percentile class

i = the class interval of the percentile class.

The confidence limits are computed by

$$w_a = k_a + i_a(A - C_a)/V_a \qquad (6)$$

$$w_b = k_b + i_b(B - C_b)/V_b \qquad (7)$$

where w_a = the percentile point for the frequency A

w_b = the percentile point for the frequency B,

and k_a = the lower limit of the class interval containing w_a

i_a = the class interval of this class

* Weatherburn (1947).

C_a = the cumulative frequency up to but not including the class containing w_a

V_a = the frequency of the class containing w_a;

k_b, i_b, C_b, and V_b are defined the same way with reference to w_b. (Note that it is possible that k_a or k_b may equal k, and so on. This does not affect the computation.)

In summary, the steps for computing w, w_a, and w_b for a frequency distribution are performed in the following order. The designation of the desired percentile point is W.

a. From the given frequency distribution, set down the class limits (exactly) and the cumulative frequency distribution.
b. Compute WN and determine, by inspection, k, i, C, and V.
c. Compute σ_{WN} according to (2).
d. Compute A according to (3), and determine k_a, i_a, C_a, and V_a.
e. Compute B according to (4), and determine k_b, i_b, C_b, and V_b.
f. Compute w, the percentile point in question, according to (5).
g. Compute w_a, the lower 2σ confidence limit, according to (6).
h. Compute w_b, the upper 2σ confidence limit, according to (7).

(The foregoing lends itself readily to machine computation, especially when, as in the present paper, the class intervals, i, i_a, and i_b, are always 1. A program has been developed for an electronic, desk size computer by one of the authors, B. L. M.)

SUMMARY

One of the epidemiologic characteristics of a disease is its durations pattern. Not only the mean duration will vary from one diagnostic entity to the next, but so will the shape of the distribution of durations, which can be described in several ways. In this paper a picture is presented for each of a number of specific diseases. For example, graphs are shown for 107 cases of measles, 368 cases of infectious hepatitis, 446 cases of mumps, 879 cases of pilonidal cyst and 2177 cases of streptococcal sore throat. These and other charts illustrate the techniques of analysis and presentation, from the point of view of the statistician, the epidemiologist, and the surgeon or hospital commander.

The curves are "cases-remaining" curves, which are strictly analogous

to survivorship curves commonly given with life tables. A cohort begins its episode of illness at the zero point on a scale of days, and diminishes as each case terminates. The cases-remaining curves are more useful than ordinary frequency polygons (on a scale of duration). They show at a glance the range and skewness of the duration distribution, the modal day for discharge from medical care, and the median and other percentile points on the "days-after-admission" scale.

The Air Force medical data are unique in that they provide unusually large frequencies, derived from uniform records and recording procedures, with diagnoses established by a physician in each case. In this presentation, the beginning of an illness is excusal from military duty due to illness, and the end is return to duty recommended by a medical officer; cases not returned to duty (deaths or disability separations from the service) are not included.

A technical note describes the method of computing confidence intervals for percentile points.

Inquiry or expression of interest, regarding any of the durations curves now on hand, will be welcomed by the Biometrics Division, Office of the Surgeon General, Headquarters, U. S. Air Force, Washington, D. C.

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SEQUENCE OF EPIPHYSEAL UNION IN A PREHISTORIC KENTUCKY POPULATION FROM INDIAN KNOLL

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INTRODUCTION

THE time and order of epiphyseal union has been extensively studied during the past 35 years by means of both post-mortem examinations and roentgenographic methods. The result of these observations has been the establishment of a sequence of union that, in spite of not being definitely fixed, can be utilized in human identification, growth research, and clinical treatment.

However, almost without exception, all of these studies have been of modern white populations, and data concerning this phenomenon in other racial groups are scarce. For this reason, the present study was undertaken. Dealing with a primitive, prehistoric series of American Indians from what is probably the largest racially homogeneous population in North America, it sets forth an age and order of union, within the limits imposed when dealing with undocumented material. This order of union is compared to other investigators' sequences and the similarities and differences noted.

The author wishes to express his gratitude to Dr. Charles E. Snow, University of Kentucky, whose advice was often sought during the research, to Gloria Enrile Punzalen, D. M. D., Philadelphia, and Joe W. Daugherty, D. M. D., Lexington, Kentucky, who assessed the degree of tooth formation, and to Dr. Herman E. Spivey, formerly Chairman of the University of Kentucky Research Fund Committee, whose grant-in-aid helped to make the research possible.

THE SERIES

Indian Knoll is one of the most fruitful archeological sites, in terms of skeletal material, ever to be excavated in North America. Over the

years, more than 1234 burials have been removed from this large shell mound in western-central Kentucky. The most comprehensive investigation was made by the University of Kentucky under the auspices of the WPA (Webb, 1946) when the remainder of the site was almost completely excavated and approximately 900 identifiable burials were found. In addition, some 70 skeletons are located at the U. S. National Museum in Washington, D. C. (Snow, 1948).

The antiquity of the site has been set at about 5302 years, by means of radiocarbon dating (Libby, 1955). The archeological remains indicate that Indian Knoll belongs to the Eastern Archaic period of American prehistory; polished stone artifacts are found, but agriculture apparently was unknown.

Snow (1948) described the skeletal material and concluded that the people morphologically belonged to the Amerindian subdivision of the Mongoloid race, Neumann's Iswanid (see Neumann, 1952). An additional demographic analysis has been made by Johnston and Snow (n. d.), using more recent methods of age determination.

METHODOLOGY

After an examination of the University of Kentucky's skeletal catalogue on Indian Knoll, some 90 burials were selected for this study. As chronological aging was accomplished by means of an assessment of mandibular third molar calcification from lateral x-rays, it was necessary to choose those where this tooth was preserved and the final series consisted of 35 males and 27 females.

Skeletal maturation was observed at 33 locations, consisting mainly of epiphyseal-diaphyseal union. Table 1 lists the areas observed. The degree of union was recorded as follows: stage 1, no union; stage 2, partial union; stage 3, completed union, all traces of fusion having disappeared. The data are presented in this manner.

Chronological ages have been assigned on the basis of the degree of calcification of the mandibular third molar, which, in the period of greatest epiphyseal activity undergoes the greatest formative changes. Both Demisch and Wartman (1956) and Lewis and Garn (1960) have found significant correlations between tooth formation and chronological age. The earlier study indicates values of +.83 for males, +.73 for females, and +.79 overall for the mandibular third molar, while the latter authors have said that, in spite of an undetermined relationship

between the two variables, tooth formation is not unusually faster or slower when growth is advanced or retarded.

Lewis and Garn (1960) have found also that, even though correlations between skeletal age and tooth calcification increased as the period of adolescence was entered, the values were still overall rather low. (For example, at 9 years, the correlation based on Greulich and Pyle, 1959, was —.305.) The high correlation between tooth calcification and chronological age, coupled with a more independent relationship between dental formation and skeletal maturation noted by these authors is felt to justify the use of this criterion in the assessment of age. Lewis and Garn have said:

> tooth formation proved no more variable and often less variable than the age-developmental variability of menarche, tooth eruption, hand age, or the appearance of ossification centers. Under these circumstances we believe that tooth formation affords a more definitive and useful estimate of chronological age in children of white ancestry when this is not known. (*ibid.*, p. 72)

Table 2 shows the 7 stages of tooth calcification utilized by Demisch

TABLE 1

List of epiphyses and anatomical areas observed

Upper Extremity	*Vertebra and Pelvis*
Humerus, Proximal Extremity	Fifth Lumbar Superior Ring
Humerus, Distal Extremity	S-1 Superior Ring
Humerus, Medial Epicondyle	S 1-2 Joint
Ulna, Proximal Extremity	S 2-3 Joint
Ulna, Distal Extremity	S 3-4 Joint
Radius, Proximal Extremity	S 4-5 Joint
Radius, Distal Extremity	Sacrum, Lateral Joints
First Metacarpal	Sacrum, Auricular Epiphysis
	Acetabulum, Primary Elements
Lower Extremity	Iliac Crest
Femur, Head	Ischial Tuberosity
Femur, Greater Trochanter	Ischial Ramus
Femur, Lesser Trochanter	
Femur, Distal Extremity	*Other*
Tibia, Proximal Extremity	Scapula, Acromial Process
Tibia, Distal Extremity	Scapula, Glenoid Fossa
Fibula, Proximal Extremity	Sternum, Clavicular Notch
Fibula, Distal Extremity	Sternum, Costal 1 Notch
Calcaneum	

and Wartman and the corresponding stages used in this study. Since it was desirable to have increased differentiation in the upper limits of the scale, where root formation is complete, it was decided to include closure of the basal suture as an additional criterion, making a 9-stage scale of development. Closure of this suture has long been recognized as an excellent indication of adulthood because of its stability relative to chronological age. (Stewart, 1952, p. 54; Cobb, 1952, p. 799; McKern and Stewart, 1957, p. 34). Only two individuals were found with closed basilar sutures and tooth formation in stage 7 and, since they were both females, they were placed in stage 7, as it is believed that they represent accelerated skeletal growth on the part of the female.

TABLE 2

Calcification stages of Demisch and Wartman (1956) and corresponding stages at Indian Knoll

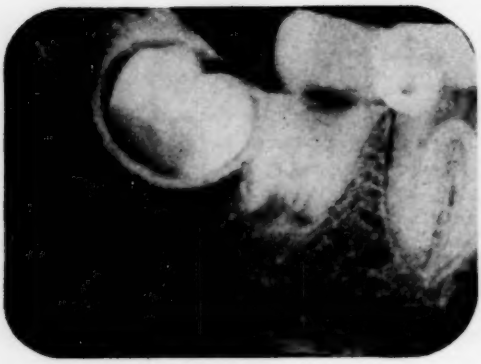
Demisch and Wartman	Indian Knoll
0. No change in bone density; no crypt.	
1. Crypt visible; no calcification.	
2. Calcification of tips of one to four cusps.	
3. Coalescence of two or more centers.	
4. Outline of cusps completed, progressing toward level of grooves	1. Crown 1/4 Completed.
5. Half crown completed.	2. Crown 1/2 completed.
	3. Crown 3/4 completed.
6. Crown completed.	4. Crown completed.
7. Beginning root formation.	5. Root 1/4 completed.
	6. Root 1/2 completed.
	7. Root 3/4 completed.
	8. Root completed, basal suture open.
	9. Root completed, basal suture closed.

Independent assessments of the degree of tooth calcification were made by Drs. Punzalen and Daugherty. Identical assessments were made in 42 cases; they differed by one stage in 19 cases and by two stages in only one case. The frequency of agreement was 68%, somewhat lower than Demisch and Wartman's figure of 78%. Examples of each stage of formation may be seen in figure 1.

Chronological ages were assigned to each of the stages of calcification

STAGE 1
#361 MALE

STAGE 2
#563 FEMALE



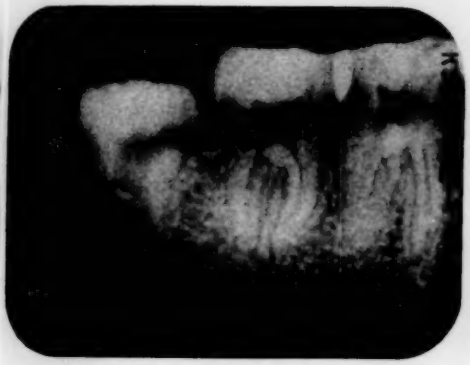
STAGE 3
#399 MALE

STAGE 4
#723 FEMALE

FIG. 1. MANDIBULAR THIRD MOLAR X-RAYS SHOWING EXAMPLES OF STAGES OF CALCIFICATION AT INDIAN KNOLL.

STAGE 5
#439 MALE

STAGE 6
#751 FEMALE



STAGE 7
#804 MALE

STAGES 8 AND 9
#769 MALE

FIG. 1 (Continued).

using the findings of Demisch and Wartman. Their median ages were, however, adjusted to compensate for the earlier ages of tooth eruption observed among American Indians (Hrdlička, 1908; Stewart, 1934; Steggerda and Hill, 1942). Half-years were used to indicate the central tendency within a range that is not definitely known. (Even Demisch and Wartman do not indicate the range of variation in their study.) However, it is felt that this variation is not extreme in the Indian Knoll group because of the homogeneity of the series in terms of morphology. Steele (1948) has commented upon this, stating that the many similarities, together with the low statistical variability found by Snow (1948), leads to the conclusion that "the basis of these things was similarity in genetic makeup." These ages are given in table 3.

TABLE 3

Median age of stages found by Demisch and Wartman (1956); corresponding ages used for Indian Knoll, and distribution of Indian Knoll series

AMERICAN WHITES (Demisch and Wartman)			KENTUCKY INDIANS			
STAGE	AGE IN YEARS		STAGE	AGE IN YEARS *	NUMBER	
	M	F			M	F
4	11.7	11.5	1	younger than 11	2	1
5	12.1	13.6	2	11.5	0	2
			3	12.5	2	0
6	14.0	12.9	4	13.5	1	3
7	14.7	14.1	5	14.5	1	4
			6	15.5	1	6
			7	16.5	6	9
			8	17.5	11	4
			9	older than 18	3	6

* Mid-point of the age group.

RESULTS

The results of the observations are given in table 4. In determining the age of union of a given epiphysis, the ages at which 50% indicated beginning and final fusion were used as the points where union could be said to have occurred in the majority of cases. However, due to the small number of individuals in some stages, it was necessary to use qualitative reasoning in some cases. The overall age-order sequence is shown in table 5.

TABLE 4

Summary of observations

CALCIFICATION STAGE	EPIPHYSEAL UNION STAGE ♂ n	1	2	3	♀ n	1	2	3
	Humerus, Proximal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	3	1	0
6	1	1	0	0	6	3	3	0
7	5	3	2	0	9	4	5	0
8	11	7	4	0	4	1	3	0
9	3	0	1	2	6	1	4	1
	Humerus, Distal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	0	1	1
3	2	2	0	0	0	0	0	0
4	1	0	1	0	3	0	3	0
5	1	0	1	0	3	0	0	3
6	1	0	0	1	6	0	0	6
7	6	0	1	5	9	1	0	8
8	11	0	2	9	4	0	0	4
9	3	0	0	3	6	0	0	6
	Humerus, Medial Epicondyle							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	3	1	0	2
6	1	1	0	0	6	2	1	3
7	6	3	1	2	9	2	2	5
8	11	4	1	6	4	0	2	2
9	3	0	0	3	6	0	0	6
	Ulna, Proximal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	0	1	1
3	2	2	0	0	0	0	0	0
4	1	0	1	0	3	0	3	0
5	1	1	0	0	3	0	0	3
6	1	0	1	0	6	0	1	5
7	6	0	3	3	9	0	0	9
8	11	4	1	6	4	0	2	2
9	3	0	0	3	6	0	0	6

CALCIFICATION STAGE	EPIPHYSEAL UNION STAGE ♂ n	1	2	3	♀ n	1	2	3
	Ulna, Distal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	2	2	0	0
6	1	1	0	0	6	6	0	0
7	6	4	2	0	9	4	5	0
8	10	10	0	0	4	3	1	0
9	3	2	0	1	6	1	0	5
	Radius, Proximal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	0	1	1
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	3	0	2	1
6	1	1	0	0	6	1	2	3
7	6	4	2	0	9	4	5	0
8	11	3	8	0	3	0	3	0
9	3	0	0	3	6	0	1	5
	Radius, Distal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	4	4	0	0
6	1	1	0	0	6	3	3	0
7	6	4	2	0	9	4	5	0
8	10	7	3	0	4	1	2	1
9	3	0	2	1	6	1	5	0
	First Metacarpal							
1	1	1	0	0	0	0	0	0
2	0	0	0	0	1	1	0	0
3	2	2	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0
5	1	1	0	0	3	0	1	2
6	0	0	0	0	5	1	1	3
7	5	2	1	2	4	0	0	4
8	6	2	1	3	1	0	0	1
9	2	1	0	1	6	0	0	6

TABLE 4 (Continued)

CALCIFICATION STAGE	EPIPHYSEAL UNION STAGE ♂				♀			
	n	1	2	3	n	1	2	3
	Calcaneum							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	1	0	1	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	2	1	0
5	1	1	0	0	4	0	3	1
6	0	0	0	0	6	0	3	3
7	4	0	2	2	7	0	4	3
8	9	0	6	3	4	0	2	2
9	3	0	0	3	4	0	0	4
	Femur, Head							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	0	1	0	3	2	1	0
5	1	1	0	0	4	0	4	0
6	1	1	0	0	6	1	5	0
7	6	2	3	1	9	1	8	0
8	11	3	8	0	3	0	3	0
9	3	0	2	1	6	0	4	2
	Femur, Greater Trochanter							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	0	1	0	3	1	2	0
5	1	1	0	0	4	0	2	2
6	1	1	0	0	6	1	2	3
7	6	3	1	2	9	1	3	5
8	11	2	7	2	4	0	2	2
9	3	0	0	3	6	0	1	5
	Femur, Lesser Trochanter							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	1	2	1
6	1	1	0	0	6	2	1	3
7	6	3	1	2	9	0	6	3
8	11	3	4	4	4	0	1	3
9	3	0	1	2	5	0	0	5

CALCIFICATION STAGE	EPIPHYSEAL UNION STAGE ♂				♀			
	n	1	2	3	n	1	2	3
	Femur, Distal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	3	1	0
6	1	1	0	0	6	3	3	0
7	6	4	1	1	8	3	2	3
8	10	6	4	0	4	1	1	2
9	1	0	0	1	6	0	3	3
	Tibia, Proximal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	3	1	0
6	1	1	0	0	6	2	3	1
7	6	3	2	1	9	2	4	3
8	11	5	6	0	4	1	3	0
9	3	0	2	1	6	0	3	3
	Tibia, Distal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	1	3	0
6	1	1	0	0	6	2	2	2
7	6	3	1	2	9	1	4	4
8	11	4	5	2	4	0	2	2
9	3	0	2	1	6	0	0	6
	Fibula, Proximal Extremity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	1	1	0	0	0	0	0	0
4	1	1	0	0	2	3	0	0
5	0	0	0	0	3	3	0	0
6	1	1	0	0	6	3	3	0
7	6	4	0	2	8	2	4	2
8	10	5	4	1	4	1	2	1
9	3	1	1	1	5	1	1	3

TABLE 4 (Continued)

CALCIFICATION STAGE	♂ EPIPHYSEAL UNION STAGE				♀ EPIPHYSEAL UNION STAGE			
	n	1	2	3	n	1	2	3
Fibula, Distal Extremity								
1	1	1	0	0	1	1	0	0
2	0	0	0	0	1	1	0	0
3	1	1	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	3	2	1	0
6	1	1	0	0	6	2	4	0
7	6	3	2	1	9	1	4	4
8	9	2	6	1	4	0	3	1
9	3	0	2	1	5	0	4	1
Scapula, Acromial Process								
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	0	0	0	0	1	1	0	0
5	1	1	0	0	4	4	0	0
6	1	1	0	0	6	4	0	2
7	6	3	2	1	7	2	4	1
8	11	8	3	0	2	1	0	1
9	3	0	1	2	6	1	2	3
Scapula, Glenoid Fossa								
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	1	0	1
3	2	2	0	0	0	0	0	0
4	0	0	0	0	1	1	0	0
5	1	1	0	0	4	0	1	3
6	1	1	0	0	6	1	2	3
7	6	3	1	2	7	0	1	6
8	11	1	7	3	2	0	0	2
9	3	0	2	1	6	0	0	6
Fifth Lumbar Superior Ring								
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	4	0	0
6	1	1	0	0	6	1	5	0
7	6	5	1	0	7	4	3	0
8	7	6	1	0	3	1	2	0
9	3	0	3	0	6	1	3	2

CALCIFICATION STAGE	♂ EPIPHYSEAL UNION STAGE				♀ EPIPHYSEAL UNION STAGE			
	n	1	2	3	n	1	2	3
Sternum, Clavicular Notch								
1	0	0	0	0	1	1	0	0
2	0	0	0	0	1	1	0	0
3	0	0	0	0	0	0	0	0
4	1	1	0	0	0	0	0	0
5	0	0	0	0	0	0	0	0
6	1	1	0	0	2	2	0	0
7	4	1	3	0	1	1	0	0
8	4	3	1	0	0	0	0	0
9	2	2	0	0	1	0	1	0
Sternum, Costal 1 Notch								
1	0	0	0	0	1	1	0	0
2	0	0	0	0	1	1	0	0
3	0	0	0	0	0	0	0	0
4	1	1	0	0	0	0	0	0
5	0	0	0	0	0	0	0	0
6	1	1	0	0	2	1	1	0
7	4	3	1	0	1	1	0	0
8	4	2	1	1	0	0	0	0
9	0	0	0	0	1	1	0	0
Sacrum, S 4-5 Joint								
1	1	1	0	0	1	0	1	0
2	0	0	0	0	2	1	1	0
3	1	1	0	0	0	0	0	0
4	1	0	1	0	2	2	0	0
5	0	0	0	0	3	0	3	0
6	0	0	0	0	5	0	3	2
7	3	1	0	2	7	0	5	2
8	9	1	7	1	0	0	0	0
9	3	0	0	3	4	0	4	0

TABLE 4 (Continued)

CALCIFICATION STAGE	EPIPHYSEAL UNION STAGE ♂				♀			
	n	1	2	3	n	1	2	3
	Sacrum, S 1-2 Joint							
1	1	1	0	0	1	0	1	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	3	3	0	0
6	1	1	0	0	6	3	3	0
7	5	3	2	0	8	3	5	0
8	10	4	6	0	3	1	2	0
9	3	0	2	1	5	0	5	0
	Sacrum, S 2-3 Joint							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	1	1	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	0	0	0	0
5	1	1	0	0	3	1	2	0
6	0	0	0	0	6	0	5	1
7	5	3	1	1	7	1	6	0
8	10	1	8	1	3	9	3	0
9	3	0	2	1	5	0	5	0
	Sacrum, S 3-4 Joint							
1	1	1	0	0	1	0	1	0
2	0	0	0	0	2	1	1	0
3	1	1	0	0	0	0	0	0
4	1	1	0	0	0	0	0	0
5	0	0	0	0	3	0	3	0
6	0	0	0	0	5	0	5	0
7	3	0	1	2	7	1	5	1
8	9	1	7	1	2	0	2	0
9	3	0	2	1	5	0	4	1
	Acetabulum							
1	1	0	1	0	1	1	0	0
2	0	0	0	0	2	0	1	1
3	2	2	0	0	0	0	0	0
4	1	0	1	0	3	0	2	1
5	1	1	0	0	4	0	1	3
6	1	1	0	0	6	0	1	5
7	6	1	3	2	9	0	2	7
8	10	0	2	8	4	0	0	4
9	3	0	0	3	6	0	0	6
	Iliac Crest							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	3	3	0	0
5	1	1	0	0	4	4	0	0
6	1	1	0	0	6	4	2	0
7	6	4	2	0	9	5	4	0
8	10	7	3	0	4	2	2	0
9	3	0	2	1	5	0	5	0

CALCIFICATION STAGE	EPIPHYSEAL UNION STAGE ♂				♀			
	n	1	2	3	n	1	2	3
	S-1 Superior Ring							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	3	1	2	0
6	1	1	0	0	5	1	4	0
7	6	3	3	0	8	5	3	0
8	8	5	2	1	3	0	3	0
9	3	0	3	0	5	0	4	1
	Sacrum, Lateral Joints							
1	1	1	0	0	1	0	1	0
2	0	0	0	0	2	0	2	0
3	2	2	0	0	0	0	0	0
4	1	0	1	0	1	1	0	0
5	1	1	0	0	3	0	3	0
6	1	1	0	0	5	0	4	1
7	5	1	4	0	7	0	4	3
8	10	1	6	3	3	0	3	0
9	3	0	1	2	5	0	3	2
	Sacrum, Auricular Epiphyses							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	3	3	0	0
6	1	1	0	0	6	5	1	0
7	7	4	2	1	8	7	1	0
8	10	8	2	0	3	1	2	0
9	3	1	1	1	5	1	4	0
	Ischial Tuberosity							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	4	3	1	0
6	1	1	0	0	6	2	4	0
7	5	3	2	0	9	1	8	0
8	10	9	1	0	4	1	3	0
9	3	0	2	1	6	0	5	1
	Ischial Ramus							
1	1	1	0	0	1	1	0	0
2	0	0	0	0	2	2	0	0
3	2	2	0	0	0	0	0	0
4	1	1	0	0	2	2	0	0
5	1	1	0	0	4	4	0	0
6	1	1	0	0	5	5	0	0
7	6	5	1	0	7	4	3	0
8	10	9	1	0	3	3	0	0
9	3	0	2	1	2	2	0	0

TABLE 5

Age and order of epiphyseal union at Indian Knoll

	FEMALE		MALE	
	Begin	Complete	Begin	Complete
Acetabulum	11.5	14.5	14	17
Humerus, Distal Extremity	11.5	15.5	14	16
Ulna, Proximal Extremity	11.5	17	14	17
Sacrum, S 3-4	12	—*	16.5	—
Radius, Proximal Extremity	12	15.5	17.5	19
Femur, Greater Trochanter	13.5	17	17	18
Calcaneum	14.5	18	16.5	18.5
First Metacarpal	14.5	15	17	18
Sacrum, S 4-5	14.5	—	17	19
Sacrum, Lateral Joints	14.5	—	17	18.5
Scapula, Glenoid Fossa	14.5	15	17	19
Femur, Head	14.5	—	17	—
Femur, Lesser Trochanter	15	18	17	18
Sacrum, S 2-3	15	—	17	—
Tibia, Distal Extremity	15	18	17	18
Fibula, Distal Extremity	15	—	17	—
Humerus, Medial Epicondyle	15	16	17	18
S-1 Epiphyseal Ring	15	—	17.5	—
Ischial Tuberosity	15.5	—	18.5	—
Tibia, Proximal Extremity	16	18	18	—
Fibula, Proximal Extremity	16	19	18	—
Humerus, Proximal Extremity	16	—	18.5	—
Scapula, Acromion	16.5	18	18.5	19.5
Sacrum, S 1-2	17	—	18.5	—
Fifth Lumbar Ring	17	—	18.5	—
Radius, Distal Extremity	16	—	18.5	—
Femur, Distal Extremity	17	18	18.5	—
Ulna, Distal Extremity	18	19	18.5	—
Sacrum, Auricular Epiphysis	18	—	18.5	—
Iliac Crest	17.5	—	18.5	—
Sternum, Clavicular Notch	—	—	18.5	—
Sternum, Costal 1 Notch	—	—	—	—
Ischial Ramus	—	—	—	—

Note: the table is organized on the basis of the beginning order in females.

* No conclusive data as to age of union.

DISCUSSION AND COMPARISON

It can be seeen from table 5 that the female shows union of the epiphyses about two years earlier than the male. This has been demonstrated many times before and the earlier maturation of the female is an established fact in all racial groups.

The works of four authors were used in comparing the order of union found at Indian Knoll: Todd's (1930) Brush Foundation series of American whites; Stewart's (1934) study of American Indians and Eskimos; Flecker's (1942) report on epiphyseal union in Australian whites; Lurie, Levy, and Lurie's (1943) work with American white children.

Rank-order correlation coefficients were computed in order to indicate the degree of relationship between the sequence found at Indian Knoll and each of the other studies. These are as follows:

American whites (Lurie *et al.*)	+.781
Australian whites (Flecker)	+.853
American whites (Todd)	+.884
American Indians and Eskimos (Stewart) . . .	+.941

The highest relationship exists with Stewart's series of prehistoric Indian and Eskimo material, a skeletal series made up of representatives of several geographical areas of North America and including a few from Indian Knoll. However, these differences were not found to be statistically significant, Lurie's sample showing significance at the .10 level, with Flecker's and Todd's samples being considerably less. Even though higher correlations were found with another American group, racial differences cannot be postulated strictly on this basis.

The ages for epiphyseal union were compared to those published by eight other authors. Table 6 shows this comparison. It can be seen that there is a range of opinion in many cases, even though all series are of white background, except for the Indian Knoll sample. The ages for epiphyseal union within the Indian Knoll group fall within this range with no apparent trend toward either early or late fusion. In one of the few studies of ossification and epiphyseal union in a non-white group, Abbey and Adey (1955) found that the ages of union among Australian Aborigines are also within the European range and even on the earlier end of the range.

TABLE 6

Ages of epiphyseal union by various authors

AUTHOR	FEMALE (1)	(2)	(3)	(5)	(6)	(8)	MALE (2)	(3)	(4)	(5)	(6)	(7)	(8)
Epiphysis													
Acetabulum	—	13	12	13	—	11.5	14–15	14	—	14	—	—	14
Humerus, Distal	—	—	13–14	13	—	11.5	—	16	14–15	14	—	—	14
Ulna, Proximal	—	13–14	14	13	—	11.5	15–16	16	—	15	—	—	14
Radius, Proximal	14	13–14	14	13	—	14	16	16	16	15	—	—	17.5
Calcaneum	14	13	14	13	—	14.5	15–16	17	—	14	—	—	16.5
First Metacarpal	12	14	15	15	14	14.5	16	18	16–17	16	15–16	—	17
Femur, Head	—	14	14	17	—	14.5	16	17	—	17	—	—	17
Femur, Greater Trochanter	—	14	14	17	—	13.5	16	18	—	17	—	—	17
Tibia, Distal	—	14–15	14	15	—	15	16–17	17	—	16	—	—	17
Humerus, Med. Epicondyle	—	13–14	14	14	—	15	16	16	16–17	15	—	—	17
Ischial Tuberosity	—	21	—	—	—	15.5	20	—	—	—	—	19	18.5
Fibula, Distal	—	14–15	15	15	—	15	16–17	17	—	16	—	—	17
Fibula, Proximal	—	—	17	18	15–16	16	19	18	—	—	18	17–18	18
Fifth Lumbar Sup. Ring	—	—	—	—	—	17	—	—	—	—	—	19	18.5
Humerus, Proximal	—	—	17	19–20	—	16	18	20	—	—	—	17–18	18.5
Radius, Distal	—	16	18	—	16–17	16	17–18	19	18–19	—	18–19	17–18	18.5
Femur, Distal	—	—	17	18	—	17	—	19	—	18	—	17–18	18.5
Tibia, Proximal	—	—	14	18	—	16	18	18	—	—	—	17–18	18
Ulna, Distal	—	16	17	18	15–16	18	17–18	19	18–19	18	17	17–18	18.5
Scapula, Acromion	—	—	17	—	—	16.5	—	17	—	—	—	18	18.5
S-1 Superior Ring	—	—	—	—	—	15	—	—	—	—	—	17	17.5
Iliac Crest	—	18	21	—	—	17.5	18	21	—	—	—	17–18	18.5
Sacrum, Auricular Ep.	—	—	—	—	—	18	—	—	—	—	—	17–18	18.5

(1) Harding, 1952
(2) Lurie et al., 1943
(3) Flecker, 1942
(4) Sidhom and Derry, 1931
(5) Todd, 1930
(6) Greulich and Pyle, 1959 (hand and wrist)
Pyle and Hoerr, 1955 (knee)
(7) McKern and Stewart, 1957
(8) Indian Knoll

CONCLUSIONS

From this study of epiphyseal union in a population of early prehistoric American Indians, the following conclusions may be drawn:

1) The female at Indian Knoll matured skeletally about two years earlier than the male. This precocity on the part of the female has been demonstrated many times in other populations.

2) The correlation, with respect to sequence of union, between Indian Knoll and three studies of white children (Todd, 1930; Flecker, 1942; Lurie, Levy, and Lurie, 1943) is significantly high. The highest correlation, however, was found with a sample of North American Indians and Eskimos (Stewart, 1934), but the differences approached statistical significance in only one case. Though racial differences may be suggested, the lack of statistical evidence to support this prohibits any inference along these lines.

3) The ages of union of the epiphyses at Indian Knoll fall within the white range.

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BOOK REVIEWS

Photography in Medicine. By Arthur Smialowski and Donald J. Currie. xviii + 330 pp. Charles C. Thomas, Springfield, Illinois, 1960. $14.50.

Photographs are often taken in the context of medical investigation and practice, particularly as records of treatment, research documents, aids in teaching, or as illustrations for publication. Some medical centers have a well organized department of photography. In fact, Mr. Smialowski, the senior author of this volume, is in charge of such a department at St. Michael's Hospital, Toronto. Dr. Currie is a surgeon in the same institution.

This book is attractive in format and broad in coverage. It tells how to set up the photographic services in a hospital, and how to use cameras and film in a wide diversity of clinical and research situations. Its chapters are mainly subdivided according to the needs of different specialties of clinical medicine and surgery.

Apart from this clinical emphasis, the book contains valuable advice for a considerable spectrum of human biologists, such as specialists in gross or microscopic anatomy, genetics or behavior. The geneticist, for example, can learn to photograph the eye in several different ways if he wishes to study familial resemblances of its many hereditary characteristics. Enough is included on infrared techniques so that he can record patterns of superficial veins, as Spuhler has done (cf. Laughlin, W. S., and S. L. Washburn, eds. 1951 Papers on the Physical Anthropology of the American Indian. Fourth Viking Fund Seminar [1949], The Viking Fund, Inc., New York, N. Y., pp. 177-202).

Other possibilities are endoscopic techniques to record many inaccessible locations such as the interior of a cranium. Chapters are included on photomicrography and cinematography, and the reader can learn how to make lantern slides, and how to prepare images and records for publication such as radiographs, charts or tables.

Despite its breadth, the book skimps on the photography of the human body for purposes such as body typing, estimations of somatic volume and surface area, and photogrammetry in general. The reader can make a start on this literature from the following references, which will put him on the track of several additional and important earlier papers:

Björn, H., C. Lundquist, and P. Hjelmström 1954 A photogrammetric method of measuring the volume of facial swellings. J. Dent. Res., *33*: 295-308.

Geoghegan, B. 1953 The determination of body measurements, surface area and body volume by photography. Am. J. Phys. Anthrop., *11*: 97-119.

Hertzberg, H. T. E., C. W. Dupertuis, and I. Emmanuel 1957 Stereophotogrammetry as an anthropological tool. Photogrammetric Engineering December 1957: 942-949.

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Some of the illustrations in *Photography in Medicine* are not entirely satisfactory. Figs. 5, 6, 7, 107 and 108, which were intended to show the utility of various backgrounds for the nude body, are unfortunately accompanied by changes in the posture and lighting of the model, so that the effects of changing one factor alone are not clearly brought out. In fig. 65, where metric rulers are illustrated for defining the scale of a photograph, the millimeter graduations are poorly drawn and inaccurately located.

These details are minor flaws in a work which in general is an eminently practical guide to many of the photographic problems of the human biologist, even if it does not anticipate every situation which may arise in his work.

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Respiratory Physiology and Its Clinical Application. By JOHN H. KNOWLES. 256 pp. Harvard University Press, Cambridge, Massachusetts, 1959. $5.25.

Striking advances in respiratory physiology over the past two decades, together with the increasing medical importance of chronic pulmonary disease, are beginning to attract human biologists other than physiologists and clinicians to this important field. As the population ages, the prevalence of chronic pulmonary disease rises steeply. Alarming increases in lung cancer and in air pollution have been well publicized, but few are aware that recorded deaths from such diseases as emphysema, while

still relatively infrequent, are mounting faster in the United States than any other cause of death. R. F. Loeb's prediction that chronic pulmonary disease would become clinical medicine's most challenging problem seems to have come true almost overnight.

The causes of asthma, chronic bronchitis ("the English disease"), and emphysema, like those of cancer and cardiovascular disorders, are multiple and poorly understood or unknown, and therefore well suited to epidemiological investigation. Studies like those of Doll on cancer and of Keys and Morris and their associates on coronary artery disease are appearing on chronic pulmonary disease, pre-eminently from the Pneumoconiosis Research Unit of the Medical Research Council in Great Britain.

Such physiologists as Comroe, Fenn, and the Nobel laureates Cournand and Richards have devised new tools for analyzing normal respiration, delineating functional abnormalities, and quantifying the progress of disease or response to therapy. Using these techniques, Brožek (J. Gerontol., *15*:155, 1960), Shock, and other gerontologists have extended our knowledge of the normal aging process.

Physical anthropologists, too, have much to contribute. Marked racial differences in the incidence and course of pulmonary infection, bacterial and mycotic, deserve systematic investigation, as G. D. Williams did in 1939 for tuberculosis (American J. Hygiene, *29*:61), while possible racial variations in normal pulmonary function have not yet been explored. Body dimensions are used to calculate not only anatomical values, like lung volumes, but many physiological parameters as well, such as vital capacity, diffusing capacity, maximum breathing capacity, and several mechanical aspects of breathing. Chest circumference and expansion, and that old anthropometric standby, the thoracic index, are functionally relevant to senile emphysema and the aging lung, while changes in the thoracic spine, as in kyphoscoliosis or senile kyphosis, influence pulmonary form and function. Many of the currently accepted values and formulas, based on limited data, could surely be improved.

For a brief introduction to respiratory physiology, Richards' 9 pages in Cecil-Loeb's *Textbook of Medicine* (Saunders, 1960) are recommended. Basic concepts and methods are treated more fully in *The Lung* (Year Book Publishers, 1955), a masterpiece of lucid exposition by Comroe, Forster, DuBois, Briscoe, and Carlson.

Knowles' book amplifies that of Comroe *et al.* in providing an excellent systematic account of the disease syndromes, in 12 chapters rather than 10 individual case studies, and a more complete and up-to-date bibli-

ography of 426 rather than 100 items. Addressed primarily to clinicians, it can be read with equal profit by many other workers, particularly those concerned with disease definition—a crucial matter in group or international comparisons. The book has two main divisions, physiological and clinical. Each physiological parameter—pulmonary ventilation, the various lung volumes and capacities, distribution of inspired air, perfusion and diffusion, the work of breathing, arterial blood analysis, and acid-base balance—is discussed in terms of principles, methods of testing, normal values and variations, use, and limitations. The clinical portion begins with a chapter on pathological physiology in pulmonary disease, with useful summary tables, and proceeds to emphysema, both senile and obstructive, asthma, heart disease, neuro-muscular disorders of respiratory function, primary and secondary polycythemia, and several other conditions, including pregnancy. For each disorder, alternative theories of pathogenesis are fairly and critically presented, and principles—though not details—of therapy are outlined, all based on the physiological principles previously expounded.

This volume will serve as a useful reference work for years to come, containing enough solid factual material for all but the most specialized investigator. In a clear, literate style that makes for pleasant reading, Knowles has simplified a difficult area, making available, for the first time in a single source, information on pulmonary function tests and their practical applications.

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Modern Problems in Pediatrics. V: Child Development. Edited by FRANK FALKNER. ix + 237 pp. S. Karger, New York, 1960. $14.00.

This book, published simultaneously as a supplement to the International Review of Pediatrics, contains discussions of the concepts and methods underlying the eight multi-disciplinary growth studies of the International Children's Center (I. C. C.). The I. C. C. is a coordinating body for growth studies now being conducted at Brussels, Dakar, Kampala, London, Louisville, Paris, Stockholm, and Zurich.

The concept of an investigative base-line underlying all of the studies is outlined in the introductory chapter, "The History and Conception of the Coordinated Studies," by Frank Falkner. According to Falkner, "The main aim of the whole coordinated research is to produce . . . timely, reliable, truly comparable data on many diverse problems of growth and development."

All of us who have been concerned with the problem of population comparisons are aware that data obtained by one investigator usually cannot be accurately compared with that reported by others. This lack of comparability may be due to a number of factors, but most commonly results from differences in measurement programs and measurement techniques. The I. C. C. endeavors to attain, in this book and through regular meetings, rigorous comparability for all the participating groups.

Dealing with "General Considerations," Colin Hindley spells out the desirability and problems of longitudinal growth studies. This excellent article is marred by a number of irritating typographical errors.

In the chapter on "The General Headings Forming the Base-Line," Frank Falkner briefly outlines the various aspects that are covered. Each is discussed in greater detail in the other chapters and in the appendices.

Marcel Graffar points out the strong influence of social environment on growth, and the need to take these factors into consideration in human growth studies. The criteria proposed seem generally suitable to European and American studies, but their applicability to non-Western cultures is very doubtful. In this connection, it is of interest, that a greatly modified form is used in the Dakar study.

Colin Hindley briefly reviews psychological theories of personality formation, points out the value of longitudinal cross-cultural studies of personality development, and discusses at some length the interview and testing techniques of the I. C. C. studies.

Frank Falkner presents in detail the techniques of making the physical measurements of the I. C. C. baseline, including tissue growth, with special reference to longitudinal studies.

Jean Sénécal recounts some of the problems of applying the I. C. C. baseline to non-European areas. Sénécal's comments are drawn from his experiences in Dakar, but seem generally applicable to similar work in other areas. To the anthropologist, it is obvious that some anthropological training—particularly on the concept of culture—would be a tremendous aid to all workers dealing with non-European groups.

In the chapter on "The Effects of Malnutrition on the Growth of

Young Children," R. F. A. Dean presents results of investigations on kwashiorkor and related dietary deficiency diseases of Uganda children. This article, excellent though it is, seems somewhat out of place in a book devoted to the concepts and methods of the I. C. C.

The practical problems of recording and analyzing data were discussed by Elizabeth Ferrell and Petter Kalberg. The I. C. C. group hopes that eventually all of their data will be entered on IBM cards, and presumably analysis will proceed along with data gathering.

The 100 pages of appendices reproduce various record blanks and give IBM card coding for the various portions of the I. C. C. studies. Examples are given of forms covering psychological methods, the social study, ante-natal, obstetric and neo-natal examinations, the clinical examinations, the somatic measurements, and the health history.

In summary, this is an excellent book, of great interest to all who are concerned with studies of human growth or who are considering beginning a growth study.

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Longitudinal Studies of Child Health and Development—Series II: Reports Based on Completed Case Studies. By HAROLD C. STUART AND ASSOCIATES. Supplement to *Pediatrics*, Vol. 24, No. 5, Part 11, pp. 871-974. The American Academy of Pediatrics, Evanston, Illinois. $2.00.

This report on studies carried out by the Center for Research on Child Health and Development (Department of Maternal and Child Health, Harvard School of Public Health), contains data on the monodisciplinary aspects of "The Maturity Series," involving a follow-up from prenatal period till maturity, roughly 18 years of age, for 134 children.

To the pediatrician to whom this was not previously clear these reports show again that physical growth is not a cut and dried affair of the Wetzel grid variety; variability, unpredictability, and individuality are among the striking findings. Patterns of different growth rates during the first three six-year periods of life are analyzed as to frequency of occurrence, and graphs of great practical usefulness for all concerned with growing children are presented. The most complicated and fasci-

nating part of the study still remains the promised land: the analysis of interrelationships of growth patterns with, among other, nutritional data and illness occurrences.

In this streamlined age of balanced diets and conformity all around, the occasional child shows a potential for a 4-fold deviation (2-fold in either direction) from the Recommended Daily Allowances for calories and protein, with what seems comparable rates of physical growth. Here again the mouth of the metabolically oriented student of growth is watering in anticipation of the analyses of interrelationships. In this study the protein intake of boys in the age range of 10-18 years is 15-20% above Widdowson's figures obtained in England. The significance of this discrepancy can be only speculated upon. The desirability of these high intakes is not demonstrated, since it remains to be shown that there is a direct correlation between dietary intakes and health record. The group studied unavoidably represents a self-selected group of North European whites, mainly Irish, living in Boston, and really comparable groups will probably be difficult to find anywhere.

One cannot escape feeling that the practical evaluator of growth always will have to consider such imponderabilia as family stature (referred to here as intrinsic pattern of growth), general state of health and emotional climate.

The reader of the 5th paper might be misled by the title "Pattern of illness experiences." Here we find an objective quantification of number and severity of illness that occurred during this period of study. While each child ends up with a score as a result of its encounters with infections and allergenic agents, with accidents and surgeons, one does *not* learn how the child experienced his illness. In recent years physicians again have had to realize that illness even of a purely infectious nature is not defined by the fate of the infecting agent, and the immunological mechanisms of the organism alone. Some of the scores might, on further analysis, show to have very little to do with the actual infection but much more with the child itself. The wide variability in the scores proves a point in this direction, as it is more than likely that the children encountered roughly equal opportunities to develop symptomatic illness. This brings us to one major basic difficulty in this type of study: by the time one finds the completed reports of the data published, collected over decennia, certain practical points of emphasis in the study of growth will have shifted. Broadening concepts of growth in a physiological sense include more and more new disciplines (cf. Friedrich Linneweh, ed. 1959, Die physiologische Entwicklung des Kindes. Springer, Berlin),

new aims in evaluation are formulated. Growth toward a full ability to answer the challenges of adult life needs to be evaluated. This involves more than physical growth alone. This concept increases in importance as interference with physical growth gradually diminish in severity through better prophylaxis and nutrition. Shifts in types of children's illnesses necessarily change needs for guidance by adequate data on normality and variability. For this reason the study of growth will need to be continued in a longitudinal fashion, with flexibility in the approach, integration of old and new findings, and continuous readjustment of aims and methods.

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Human Development. By PHYLLIS C. MARTIN and ELIZABETH LEE VINCENT. v + 541 pp. The Ronald Press Company, New York. 1960. $6.50.

Human Development is not the kind of book customarily reviewed in this journal, being an elementary text of the junior college, freshman sort. Several features, however, commend its notice here, among them the need for such a work, and the quality of both illustration and prose.

College students are woefully ignorant of human anatomy, nutrition, physiology in general and the physiology of reproduction in particular. These lacks are foreseeen in *Human Development* which is primarily factual human biology, and not "child development" or "human development" in the usual sense.

Most such attempts are pictorially weak, interlarded with copies of illustrations of questionable value and quality. This particular work attains a uniformly high level, with specially drawn line-cuts, with El Greco figures illustrating the digestive system and with a charmingly pony-tailed miss displaying the organs of generation. The half-tones illustrating developmental anatomy and parturition are excellent indeed.

Besides being an anatomy-physiology course in palatable capsule, *Human Development* aims higher as well, delving into man's place in nature, the antecedents of modern man, and the population problem which will shortly make kneeling in church impractical. As is the

current trend in educational manuals, there are end-of-chapter questions which students will presumably review and discuss, though with inadequate knowledge to bear.

There are, of course, precious few references, in part due to the elementary nature of the contents (what reference do you give to the functions of the liver?) and in part due to the intended level of use (beginning students are not scholars). Edna Lee Vincent, whose previous collaboration with Marion E. Breckenridge yielded 1100 references in one middle-level text, has speeded up learning by discarding the scholarly paraphernalia of footnotes, text references, *op cits*, asterisks and daggers.

This book exemplifies American technical excellence in developing education materials, almost (but not quite) proving the slogan that "learning is fun." While wondering how much the nubile reader will learn about neurons and axons, I must admit that the grim, scholarly approach is no better neurological vehicle.

To the reviewer, this text is best on the level of facts, but could well leave the realm of ideas to Medawar. Still the attempt to fence with ecology at least deserves mention. Besides, there is a very large audience, and a largely female audience, to which *Human Development* will appeal, and the authors seem to hold the pulse of their readers without peering over their own shoulders for the reactions of the scientific fraternity. In review language "this book seems eminently suitable for its particular audience," which is a wide audience indeed.

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BOOK NOTICES

DESIGN FOR A BRAIN. By W. Ross Ashby. Second Edition. ix + 256 pp. Wiley, New York, 1960. No price indicated. The search of a cybernatically oriented neurophysiologist for the explanation of adaptive behavior. The brain is treated as an organ that developed in the process of organic evolution as a specialized means of survival. The emphasis is on genetic control, of two kinds: direct (when all the regulatory mechanisms are specified *in detail* by the gene-pattern) and indirect (when the environment, through learning, fills in the details, with the gene-pattern controlling the adapting mechanisms *in general outlines*).

EXERCISE AND FITNESS. vii + 249 pp. The Athletic Institute, Chicago, Illinois, 1960. $3.00. A collection of papers presented at a Colloquium sponsored by The University of Illinois College of Physical Education and The Athletic Institute, and devoted to the effects of physical activity on fitness. The latter term was interpreted as covering both health (organic conditions) and motor performance capacity. Of special interest here are the papers on exercise and weight control (Jean Mayer), psychological correlates of fitness and physique (R. B. Cattell), changes induced by exercise in adults (T. K. Cureton), and coronary heart disease considered in reference to sedentary and physically active occupations (H. L. Taylor).

SEX DIFFERENTIATION AND DEVELOPMENT. Edited by C. R. Austin. x + 198 pp. Memoirs of the Society for Endocrinology No. 7. Cambridge University Press, Cambridge, England, 1960. $8.50. Excellent in its own right and broadly zoological framework, the volume has only limited direct relevance for human biologists (cf. integumentary sex characters of mammals, esp. of sub-human primates, p. 141; sex reversal and discrepancies between different methods of sex determination, p. 154).

DISEASE AND THE ADVANCEMENT OF BASIC SCIENCE. Edited by Henry K. Beecher. xi + 416 pp. Harvard University Press, Cambridge, Massachusetts, 1960. $12.50. A series of 21 Lowell Lectures centered around the theme, spelled out by the editor in the Preface, that some kinds of fundamental advance in science can be made only from the study of disease. Among the areas specifically considered is biochemical genetics, developmental biology, and nutrition, with reference to obesity.

ELEMENTS OF VITAL STATISTICS. By Bernard Benjamin. 352 pp. Quadrangle Books, Chicago, Illinois, 1959. $10.00. Covers the periodical population census and the ongoing registration of births, deaths and marriages, measures of fertility, mortality and morbidity; statistics of specific diseases; and uses of statistics for other purposes (hospitals, medical practitioners) than those of public health agencies. The last chapter deals

with field studies involving direct observation of disease prevalence or of social and economic (and, let's add, biological) characteristics of populations. Brief reference is made to sampling, record keeping, training of investigators, and analysis of results.

THE STRUCTURE AND FUNCTION OF MUSCLE. Edited by G. H. Bourne. Vol. 1. xvi + 472 pp. Academic Press, New York, 1960. $14.00. The first volume of the comprehensive "Muscle" trilogy, dealing with structure, mostly the microscopic structure of muscles. The first chapter, by R. D. Lockhart describes the anatomy of muscles in relation to movement and posture.

THE BIOCHEMISTRY OF DEVELOPMENT. By Jean Brachet. xii + 320 pp. Pergamon Press, New York, 1960. $10.00. Outstanding treatises in this field have been published in the English language at intervals of about a decade (J. Needham's Chemical Embryology, 1931, Biochemistry and Morphogenesis, 1942; Brachet's Chemical Embryology, 1950). The present volume is designed to acquaint the reader with recent advances and the main problems facing chemical embryologists. The developing egg, invertebrate and vertebrate, and the embryo are the focus of the monograph.

LOW-LEVEL IRRADIATION. Edited by Austin M. Brues. v + 148 pp. American Association for the Advancement of Science, Washington, D. C., 1959. $3.25. Proceedings of a symposium on the consequences of exposing human beings to low levels of ionizing radiation generated by radioactive fallout. The first part of the symposium deals with the scientific background and includes chapters on genetic (E. L. Green) and somatic (A. M. Brues) effects. The second part is concerned with the responsibility in these far-reaching matters of the press, public health authorities, statesmen, and philosophers.

LAMARCK AND MODERN GENETICS. By H. Graham Cannon. xii + 152 pp. C. C. Thomas, Springfield, Illinois, 1960. $3.75. Written on the 150th anniversary of Lamarck's *Philosophie Zoologique*, the volume aims to "tell the true story of this greatest of all French biologists" and to give a drubbing to the upholders of the neo-Mendelian mutation-selection hypothesis, as applied to the theory of evolution.

MINERAL METABOLISM: AN ADVANCED TREATISE. Edited by C. L. Comar and Felix Bronner. Academic Press, New York, 1960. Price not indicated. A comprehensive presentation of the advances made since the publication of A. T. Shohl's *Mineral Metabolism* (Reinhold, New York, 1939). This is part A of Volume 1, dealing with Principles, Processes, and Systems. J. R. Robinson's chapter (pp. 205-248) on "Body fluid dynamics" has bearing on the analysis of body composition.

PERSPECTIVES IN PERSONALITY RESEARCH. Edited by Henry P. David and J. C. Brengelmann. ix + 370 pp. Springer, New York, 1960. $7.50. The volume, published under the auspices of the International Union of Scientific

Psychology, contains contributions from 27 outstanding men representing 11 different countries. It is introduced by Henry A. Murray's informative presentation of Historical Trends in Personality Research (pp. 3-39).

MÉTHODES INDIRECTES DE MESURE DE LA GRAISSE CORPORELLE DES MAMMIFÈRES. By Bernard Louis Dumont. 67 pp. Centre National de la Recherche Scientifique, Paris, 1958. A review of methods for estimation of body fat based on determinations of body density, total body water, and subcutaneous adipose tissues. Applications to animal studies are considered.

MEN AND MOMENTS IN THE HISTORY OF SCIENCE. Edited by Herbert M. Evans. viii + 226 pp. University of Washington Press, Seattle, Washington, 1959. $4.50. Aristocratic in the genuine and precious sense of the term, the History of Science Dinner Club has existed on the Berkeley campus of the University of California since 1932. These essays, ranging widely over the *globus intellectualis*, celebrate the 25 years of existence of the club. Egon Brunswick, a psychologist, contributed an essay on "Ontogenic and other developmental parallels to the history of science."

ANATOMY: A REGIONAL STUDY OF HUMAN STRUCTURE. By Ernest Gardner, Donald J. Gray and Ronan O'Rahilly. xvi + 999 pp. Saunders, Philadelphia, 1960. $15.00. In spite of the subtitle, the approach represents—in the authors' terminology—a "moderate regionalism," a combination of the regional method of presentation with systematic considerations. While the length and the amount of detail is tailored to the needs of undergraduate courses in human anatomy, the references are cited for the benefit of the more advanced and more specialized students. Drawings are supplemented by radiographs and photographs.

AEROSPACE DICTIONARY. By Frank Gaynor. 260 pp. Philosophical Library, New York, 1960. $6.00. The biological contribution to space-travel terminology is, so far, limited and the number of specific entries, not likely to be found elsewhere is small (cf. "bioastronautics," "body burden," "space medicine"). But should you wish to check on the date of the launching of Sputnik I or to verify that "brain" in the aerospace colloquial terminology is an expression for the guidance system of a missile or unmanned rocket, Gaynor's Dictionary will serve you well.

PERSONALITY. By J. P. Guilford. xiii + 562 pp. McGraw-Hill, New York, 1959. $7.50. An upper-class textbook, oriented toward description and measurement of the individual personality: "The unifying basis of (the underlying) theory comes from a wedding of the logic of experimental method and factor analysis."

PSYCHOLOGICAL SCALING: THEORY AND APPLICATIONS. Edited by Harold Gulliksen and Samuel Messick. xvi + 211 pp. Wiley, New York, 1960. $5.00. Proceedings of a conference concerned, at a highly technical level, with the heart of the study of human behavior as a quantitative science. The

problem of scaling techniques was considered in a wide variety of contexts (decision making, psychophysics, attitude measurement, mental abilities). The topic dealt with by L. R. Tucker ("Intra-individual and inter-individual multidimensionality") clearly transcends the confines of psychometrics.

PSYCHIATRIC DICTIONARY. Third Edition. By Leland E. Hinsie and Robert H. Campbell. ix + 788 pp. Oxford University Press, New York, 1960. No price indicated. While psychiatric terminology constitutes the core of the volume, a number of entries deals with topics in the areas of constitutional medicine, psychobiology, genetics, and eugenics.

DIMENSIONS OF MIND. Edited by Sidney Hook. xiii + 281 pp. New York University Press, 1960. $5.00. Proceedings of the third annual New York University Institute of Philosophy (May, 1959), with the mind-body problem as the focal theme, including considerations of the relations between the brain and the machine.

BIOMETRIC GENETICS. Edited by Oscar Kempthorne. viii + 324 pp. Pergamon Press, New York, 1960. $8.50. The theory of quantitative inheritance is concerned with problems typically encountered by animal and plant breeders, i. e. problems in which—in contrast to situations studied in the classical Mendelian genetics—genotypes are not identifiable, and the quantitative attributes are more readily affected by variations in the environment. The participants in the Ottawa (1958) International Symposium on Biometrical Genetics discussed very competently theoretical genetics (with F. Keiter's chapter on problems of multifactorial genetics in man), design of experiments, and experimental results.

AKZELERATION UND ERNÄHRUNG. FETTLÖSLICHE WIRKSTOFFE (Growth acceleration and nutrition. Fat soluble vitamins). Edited by J. Kühnau. 98 pp. Steinkopff, Darmstadt, Germany, 1959. DM 19.00. The first two lectures, presented by W. Lenz and R. M. Du Pan at the second scientific meeting of the German Nutritional Society (pp. 1-51), deal with a matter that has been repeatedly confirmed—the secular trends in the increased body size of children—but the etiology of which continues to challenge students of human growth.

SIGERIST ON THE HISTORY OF MEDICINE. Edited by Félix Martí-Ibáñez. xviii + 313 pp. MD Publications, New York, 1960. $6.75. Perhaps some fairly pertinent chapters could be cited (e. g., the essay on the foundation of human anatomy in the Renaissance). However, the volume is registered here unashamedly for the sake of sheer delight of so many passages and essays (The Importance of Being Ernest, for one), written by a uniquely omnivorous mind examining all events *sub specie historiae*.

THE ORIGIN OF LIFE ON THE EARTH. Edited by A. I. Oparin and others. xv + 691 pp. Pergamon Press, New York, 1959. $15.00. Proceedings of the

first international symposium on the subject, held at Moscow in 1957. The topics include the primary formation and the transformation of primitive compounds; the origin of proteins, nucleoproteins and enzymes; the origin of structure; and the evolution of metabolism.

THE HEAD, NECK, AND TRUNK. By Daniel P. Quiring. 2nd edition, revised and edited by John H. Warfel. 124 pp. Lea and Febiger, Philadelphia, 1960. The volume, a companion to the author's *The Extremities* (1960), portrays in well-drawn diagrams and briefly describes the individual muscles of the region specified in the title. Reference is made to origin, insertion, and function of each muscle as well as to its nerve and artery supply.

HENRY E. SIGERIST ON THE SOCIOLOGY OF MEDICINE. Edited by Milton I. Roemer. x + 397 pp. MD Publications, Inc., New York, 1960. $6.75. The volume documents the many-sided interests of the author in the interaction between medicine and society. In the present context the chapter on the geography of disease ("Problems of Historical-Geographical Pathology," pp. 299-307) deserves to be pointed out.

WORK AND THE HEART: TRANSACTIONS OF THE FIRST WISCONSIN CONFERENCE. Edited by Francis F. Rosenbaum and Eston L. Belknap. xxiii + 537 pp. Hoeber, New York, 1959. $12.00. In this otherwise comprehensive survey of cardiovascular physiology and pathology related to physical work, only incidental and limited attention was paid to such problems as body weight and obesity.

THE ANALYSIS OF VARIANCE. By Henry Scheffé. xvi + 477 pp. Wiley, New York, 1959. $14.00. The author's aim was to elucidate in a unified way the basic theory of the analysis of variance, with heavy reliance on vector and matrix methods (to which an introduction is provided in the appendix) and a geometrical interpretation involving the resolution of observed quantities into vectors corresponding to different sources of variation. The volume is intended for a one-semester course at the senior or graduate level, and for self-study.

DER DIABETES MELLITUS ALS VOLKSKRANKHEIT UND SEINE BEZIEHUNG ZUR ERNÄHRUNG. (Diabetes as a disease of populations and its relation to nutrition). By E. G. Schenck and C. H. Mellinghoff. xi + 310 pp. Steinkopff, Darmstadt, Germany, 1960. DM 54.00. The point of departure is a unique and extensive survey of diabetic morbidity in Germany but the author pursues his subject matter, along the lines of "geographic disease studies," beyond the borders of Germany, with special reference to food intake and nutritional status.

A STRUCTURE OF SCIENCE. By Joseph H. Simons. 269 pp. Philosophical Library, New York, 1960. $4.75. A physical scientist's meditations about science. Biologists may read with some profit the first section ("Science and its Devotees"). The second part deals with general concepts, including equi-

librium, change, chance. The third part is concerned with collisions of particles.

PRINCIPLES AND PROCEDURES OF STATISTICS. By Robert G. D. Steel and James H. Torrie. vii + 481 pp. McGraw-Hill, New York, 1960. An outgrowth of courses in statistics offered at the graduate level in colleges of agriculture, with the subtitle "with special reference to biological sciences."

CRIME, JUSTICE AND CORRECTION. By Paul W. Tappan. xiii + 781 pp. McGraw-Hill, New York, 1960. $7.95. A comprehensive treatment of criminology, divided into 3 parts (Causation of crime; Administration of justice; Correction). One chapter (pp. 83-111) is devoted to physical, constitutional and hereditary factors.

SCIENCE IN PROGRESS. Eleventh Series. Edited by Hugh Taylor. xii + 379 pp. Yale University Press, New Haven, Connecticut, 1960. $7.50. Of special interest in this biennial harvest of the National Lectureships of the Society of Sigma Xi are the contributions by B. F. Skinner (experimental analysis of behavior), R. J. Williams (chemical anthropology), and E. C. Hammond (smoking and death rates).

THE PRECURSORS OF ESSENTIAL HYPERTENSION AND CORONARY ARTERY DISEASE: CHARACTERISTICS OF THE JOHNS HOPKINS MEDICAL STUDENTS. Collected papers by Caroline Bedel Thomas and others. Vol. 1, 1948-1959. x + 485 pp. The Johns Hopkins University School of Medicine, Baltimore, 1959. Price not indicated. Individuality, quantitatively assessed, is one of the fundamental concepts of human biology. Dr. Thomas and her coworkers were interested for a number of years in the relations between biological (physiological, metabolic, psychological, later also morphological) characteristics of individuals as possible precursors (predictors) and as guideposts to the prevention of essential hypertension and coronary artery disease. In this bountiful harvest are included 4 papers (pp. 41-104) concerned with the genetic aspects of heart disease.

PRAKTISCHE SPERMATOLOGIE. By H. W. Vasterling. xi + 224 pp. Thieme, Stuttgart, Germany, 1960. $6.40. Concerned with man's *potentia generandi* with focus on the physical, biochemical, morphological, and physiological features of the human semen and spermatozoa.

THE LIFESPAN OF ANIMALS. Edited by G. E. W. Wolstenholme and Maeve O'Connor. xii + 324 pp. Little, Brown and Co., Boston, Massachusetts, 1959. $9.50. The fifth and last in the Ciba Foundation Colloquia on Ageing, containing a combined index to this important series. Human biologists will find a number of papers that are directly relevant, dealing with such topics as actuarial aspects of man's life span, effects of parental age, and the onset of major diseases resulting in death.

THE HEART IN INDUSTRY. Edited by Leon J. Warshaw. xix + 677 pp. Hoeber, New York, 1960. $16.00. The volume, written by 24 authors, is clinically

oriented. The physiological effects of work on the heart are described by L. Brouha (pp. 47-104).

ANNUAL EPIDEMIOLOGICAL AND VITAL STATISTICS, 1956. 705 pp. World Health Organization, Geneva, Switzerland, 1959. $12.00. The largest part of the present volume deals with deaths according to cause, following the Abridged List (50 categories). The data are tabulated by age and sex for 52 countries. Valuable information is provided on the population and area of various countries and their vital statistics (natality, general mortality, infant mortality).

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EVOLUTION AND THE THERMODYNAMIC IMPERATIVE[1]

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THIS paper is an attempt to review some aspects of evolution in the light of the second law of thermodynamics. It is proposed that four processes common to organic and social evolution which retard the increase of entropy on this planet provide a unifying framework for the study of evolution and also have ethical consequences.

Evolution and energy transformation have been related to one-another many times previously. One postulated relationship revolves around the principle of maximizing energy transformations: Lotka (1945), for example, emphasizes the selective advantage of ecological systems with greater energy inflow and output, while White (1949) outlines a progression of human societies toward increasing use of energy. The other major relationship hinges on the apparent contradiction between the increase of organization associated with evolution and the increase of entropy predicted by the second law of thermodynamics. This law, derived from measurements made upon closed systems, gives the decrease of energy available for work or the increase of randomness accompanying physical and chemical transformations. The extrapolation of this law to the whole universe has led to Nernst's theory of "Wärmetod" or thermal death. Eddington (1929) has therefore characterized the operation of the second law as "time's arrow." It became quickly apparent, however, that organic processes result in the building up of energy stores and a decrease in the randomness of the arrangement. Needham (1943) resolved this dilemma by differentiating thermodynamic order from biological organization, while Schrödinger (1945) argued that in living matter the classical laws of physics—while not negated—are modified just as they are at extremely low temperatures. Blum (1955) has reiterated that the second law of thermodynamics does apply to bio-

[1] A shorter version of this paper was delivered at the meeting of Section H, American Association for the Advancement of Science, New York, December 30, 1960. Criticisms of a preliminary draft by Drs. Ernst Caspari and Robert March are gratefully acknowledged. If the writer persists in holding notions inadequately justified, it is clearly his sole responsibility.

chemical transformations and that the amount of energy flow from the sun during the period of evolution—and consequently the increase in entropy—has been enormous. In relative terms, however, as Johnstone (1921) has pointed out, this decrease in available energy is less than it would have been if only inorganic material had been present on the surface of the earth. Breder (1942) speculated along these lines that life as we know it may be just one of several processes retarding entropy in the universe and could conceivably contribute to a reversal of "time's arrow." It is quite possible that the second law is not applicable to open systems at all; since it implies by definition "death by confinement" (Brillouin, 1949). If the pulsating or oscillating model of the universe (see Öpik, 1960) is shown to be the most accurate, the specter of thermal death will be unfounded.

These qualifications notwithstanding, it is quite clear that the survival of life on earth is dependent on the continued availability of transformable energy. Hence the title of this paper, taken from an article by Lindsay (1959) where he proposes a "thermodynamic imperative" which "urges all men to fight as vigorously as possible to increase the degree of order in their environment so as to combat the natural tendency for order in the universe to be transformed into disorder" (p. 376). It might therefore be useful to explore again the ways in which evolution can be seen as retarding the increase of entropy. Evolution is taken here to include processes on physio-chemical, biological, ecological, and cultural levels which have taken place since the origin of biopoesis.

Persistence, Replication and Environmental Modification.

Current theories on the origin of life brought together at the 1957 International Conference in Moscow (Oparin, 1959) rely on the notion that a series of new types of chemical reactions with small chances of occurrence took place over a long period of time. Although these events were reversible, larger molecules were built up faster than they were destroyed (Gaffron, 1960). This bears a certain similarity to mutation and natural selection. The probability of a "favorable" mutation is very small, but given enough individuals and generations, its occurrence approaches certainty (Dobzhansky, 1960). Once such a mutation occurs, its chances of persistence are increased by the action of natural selection. Cultural innovations have been seen as parallel to mutations (Gerard, Kluckhohn, and Rapoport, 1956). The probability of a social innovation is much higher than that of the formation of polypeptides before there

were living cells, and the transmission of culture from one generation to the next occurs by a mechanism much more subject to variation than the intercellular transmission of genetic material in meiosis. Nevertheless, perpetuating mechanisms for social innovations show considerable regularity (Barnett, 1953). Here, then, is the first way in which entropy might be retarded in evolution: given the occurrence of an unlikely event, its continued presence may attain a higher probability than was true for the initial occurrence. This will be called the phenomenon of *persistence.*

Persistence is often accomplished by the mechanism of *replication.* Polypeptides tend to form aggregates, chromosomes are capable of autocatalysis, and culture is recreated from generation to generation. This is Schrödinger's "order from order" mechanism which he contrasts to the statistical "disorder from order" prediction embodied in Boltzmann's formulation of entropy (Schrödinger 1945:80).

Persistence and replication both amount to a modification of the system in the sense that the course of events would have been different—and tending to greater entropy—if they had not taken place. Phenomena such as environmental conditioning in ecology, inductance in embryology, and the domestication of plants and animals by man, exemplify *environmental modifications* differing from the effects of persistence and replication alone. Whatever the total effect on entropy, modification of the environment by a living organism usually decreases the chances for "disorder" from its local point of view by increasing its chances for survival as a species. Changes in the environment effected by single organisms are less striking than those resulting from aggregations and communities. Some animals build individual nests, but at the cost of considerable expenditure of energy. Populations, on the other hand, such as aggregations of mealworms, have been found to regulate their microclimate by coactions which require relatively small expenditures of energy (Allee *et al.*, 1950:362).

Does the extent of these three processes of entropy retardation show any tendency to change during the period of time encompassed by evolution? The earliest organisms probably "lived upon" a store of organic molecules accumulated in the sea (Wald, 1955). If photosynthesis had not become a fact following this period, environmental modification would have ultimately negated the effects of persistence and replication. Photosynthesis was evolved, however, and live organisms not only persisted and multiplied, but also effected further changes in the environment. The emergence of animals which do not carry on photosynthesis might again

have jeopardized what had gone on before. This did not occur either; their proliferation remains limited, and a great proportion of the vegetative material is never "consumed" at all and some of it is not even decomposed (Bates, 1960:559). When an individual organism is parasitized, eaten or killed, a great deal of energy is lost as its molecules are reassembled to fit the pattern of the organism on the next trophic level. If this loss of energy occurred at a faster rate over the whole biosphere than the "fixing" of energy by the producers, organic life should have petered out long ago. A large amount of the total protoplasm, furthermore, is replicated by cell division, thus bypassing the loss created by the death of every successive generation of individuals in multicellular reproduction. The accumulation of energy-rich deposits is the result of this "favorable balance of payments" in the organic sector.

Most of the great initial evolutionary radiations which peopled the sea took place during the Precambrian, while the occupation of land and the air was essentially completed during the Mesozoic. In general, life since the Mesozoic has been relatively stable despite the great diversification among insects in the Cenozoic (Bates, 1960). Simpson (1960), on the other hand, has pointed out that while new phyla and classes have not arisen steadily, there is apparently no diminution of differentiation on lower taxonomic levels. The reason for this is that once a primary radiation has taken place, all subsequent ones can have a much more restricted scope only. Huxley (1942), in describing this phenomenon, used the analogy of the barrel which is becoming increasingly filled with objects of smaller and smaller size. It is quite evident that this was not a gradual process. The great primary expansions were followed by temporary plateaux and even some setbacks. But, as long as the total biomass remains constant or is increasing, as long as relay radiations reoccupy most temporarily unused habitats, and as long as new niches—no matter how small—are filled, entropy retardation is continuing and probably increasing.

Structuring and Social Evolution.

Social evolution will be substituted here for Huxley's (1955:6) *human* evolutionary phase for it allows for a discussion of social factors among non-human groups together with those applicable to man alone. His statement that biological advance has terminated with the advent of culture can be questioned on the basis of the evidence from epidemiology and human genetics (*e.g.*, Motulsky, 1960). It has been noted already

that environmental modification is less striking in single molecules and organisms than in communities. In an aggregation the environment of any individual contains a number of like individuals who modify the environment in similar ways. The concept of structure implies some regular inhomogeneity in an aggregation. While many inorganic aggregations manifest structure in this sense, the amount of environmental modification they achieve is minimal when contrasted with organic molecules, cells, organisms, or communities.

Three processes of entropy retardation have been delineated, *structuring*, it seems, must be a fourth. The phenomenon of replication does not determine whether the thing being duplicated is homogeneous or not. First of all one must note that periodic organization is less probable than no organization at all. While energy is made less available in the process of crystallization, nevertheless a crystal is a less probable state of events than a solution of salt. But aperiodical organization is even less probable. Needham (1943) in discussing entropy contrasts "mixed-upness" with "separatedness." In the classic example, two vessels filled with a gas at different temperatures are "separate" and also asymmetrical; when they mingle and come to an equilibrium the system becomes symmetrical and entropy is at its maximum. Similarly, in information theory, a periodic message has little "negative entropy." The message carried by mechanical dolls performing their dance on top of a music box is minimal (Wiener, 1954). Schrödinger (1945) is making the same point when he writes: "An organism's astonishing gift of concentrating a 'stream of order' on itself . . . seems to be connected with the presence of the 'aperiodic solids,' the chromsome molecules, which doubtless represent the highest degree of well-ordered atomic association we know of—much higher than the ordinary periodic crystal . . ." (p. 77).

The proposition that structuring advances in the course of evolution was already a major element in Herbert Spencer's thought, and has been recently summarized by Huxley (1956): "Evolutionary advance is achieved through a succession of dominant or successful types, each embodying some improvement in general organization. . . . Each specialized trend shows a steady improvement in respect of its own specialization, until it reaches a limit and becomes stabilized. If a new type is evolved possessing some new improvement in general organization, it may become dominant in its turn . . ." (p. 6).

Many of the difficulties in the study of social evolution stem from the

practice of dealing with the evolution of *single* units—one species or one cultural tradition only. Thus, natural selection on the community level is rarely mentioned outside of ecological circles. Community succession is at best an approximate recapitulation of evolutionary events, but it seems significant that gradual irreversible changes have been found from slightly structured systems with rapid dynamics to thermodynamically more efficient communities made up of larger organisms (Margalef, 1958: 60). This finding may indicate that the tendency of natural systems to operate at levels of efficiency producing the maximum output (Odum and and Pinkerton, 1955) is more applicable to the times when they are becoming established—as during a period of adaptive radiation or colonization—than during their climactic phase. This hypothesis fits with Huxley's notions of social evolution quoted above. The steady improvement in the specialization of a breeding population, biotic community, or human social system might well be accompanied by improved thermodynamic efficiency. In order to reach a new adaptive peak, however, once it is established, a system could well have to traverse an adaptive trough (Wright, 1932) entailing a loss of accumulated energy reserves. This is the thermodynamic cost of "saltating" evolution.

Recently, some anthropologists too have paid considerable attention to changes taking place within systems of several interacting groups. Redfield's (1947) concept of the "width of integration" in folk societies and its elaboration by Tax (1946) for peasants were based on the consideration of inter-community as well as intra-community interactions. Childe (1951), dealing specifically with social evolution, emphasized in this context that "archeological evidence is available for inter-communal specialization. Even in Neolithic cultures we know communities of flintmakers and axe factories whose products were widely exported" (p. 63). In the same vein, Caldwell (1959) prefers to use the concept of "interaction area" to "culture area" since it is better suited to the archeological facts of continuous interareal diffusion of cultural forms. Discussing the problem of state formation, Gearing (1958) delineates a "Mesopotamian career" where "naive" states (who have no previous history of statehood) may arise through the conquest and forceful dominance of one community over others. Or these naive states may arise out of segmentary societies when ranking lineages or clans gain monopoly over functions deemed vital, for example rain-making, and through such monopoly gain coercive leverage over lesser lineages and clans. These theoretical trends have recently been brought together by

Lesser (1960). This aproach to culture history will be characterized here as *epicultural* (cf. "epicycle"—Gray, 1958, and "epiorganism"—Gerard, 1958).

Such a shift in emphasis in anthropology would correspond to a consideration of *synecology* along with *autecology*. In the latter approach the ecologist studies one species in its interactions with the total environment; in the former communities of interacting species are studied together (Allee *et al.*, 1950).

Steward's (1955) concept of "socio-cultural levels of integration" is also helpful for an understanding of differential survival of human epicultural systems. He divides a national culture, for example, into two general kinds of features: those that operate on a national level and those that pertain to given sociocultural segments of the population. In studying the evolution of such a national culture one would have to see how selection operates on the various segments, on the national level, and on the ways the segments and the national level are integrated into a total system.

When we examine human evolution in the light of the epicultural approach we can see that interaction areas between groups (and levels of integration) have become more and more extensive—until today, when much of the land-surface of the earth is part of a single ecological system united by the human species and the organisms associated with it.

Early hunters and gatherers replicated themselves slowly, occupied small and isolated territories, and probably had simple social structures. They did not modify their environment very much—moving around to find food and shelter where it was available. The building of settled villages meant a more intensive modification of the environment, eventually leading to the deliberate nurturing of animals and plants. This constituted purposeful replication of available energy sources for future use. Often agricultural settlements developed mutualistic relations with surrounding hunters, necessitating increased structuring of intergroup relations—whether warlike or amicable. Symbiotic relations between farmers and nomad pastoralists also fall into this category.

The development of trade and artisan specialists constitutes a second step away from activity directly concerned with immediate consumption of food. The potter can work at the wheel with the expectation that enough calories will be available in the coming months to feed him as well as the food producer—just as the first food producer had enough to eat while he was investing his energy for a far-away future harvest.

Human social evolution, like organic evolution, did not proceed in a straight line. If we examine the epicultural systems that were most highly developed in any period of time, we can see a progression toward larger and more complex structures which could modify their environment to greater and greater degrees. As Sahlins and Service (1960:33) have pointed out, this progression need not occur within single phylogenetic lines. Human evolution has some elements resembling the passing on of the Olympic torch from one runner to the next. Societies marginal to the climactic communities in "cultural dominance" (Sahlins and Service, 1960: Chapter 4) sometimes take over the torch of progress, as Park (1928) suggested on the basis of his marginal man theory. New torches, of course, are also lit in epicultural systems distant from other climactic communities, as in the case of Meso-America.

Increased replication of human beings (or population increase as it is usually called) and increased complexity of structuring accompanied the food-producing, urban, and industrial revolutions. The population explosion, the rapid use of accumulated energy supplies, and the disturbance of many ecological equilibria have now brought us to a point, however, where entropy retardation (and our civilization) may once again be in jeopardy (Sears, 1957). These dangers—not to mention the threat of hydrogen warfare which could increase entropy by a fantastic leap—constitute the thermodynamic imperatives of our time. Far in the future, the cooling of the sun or the contraction of the universe may present other imperatives to our descendants if they manage to survive that long.

The Thermodynamic Imperative.

Lindsay (1959) who, as previously noted, coined the expression used in the title of this essay, holds that since entropy is on the increase in the universe, we as live, conscious and human beings should fight it: ". . . the only way to make the most of life, to fulfill its purpose, if it has any, is to continue to respond to the challenge by maximizing the consumption of entropy in every possible way" (p. 385). If "purpose" can be ascribed to live organisms, it is in connection with the survival of tissues, individuals, societies, species, communities and traditions. Until now life has succeeded in maintaining itself: its persistence has retarded entropy. It has persisted by generating order from order, i.e. replication, and by modifying its environment. Replication, furthermore, has proceeded in the direction of more and more structuring, and this

structuring has accompanied an increasingly wider integration of systems until today the whole planet, in many respects, is just one single system. Could the *recognition* of this trend constitute yet another way of retarding entropy?

Rosenbluth, Wiener and Bigelow (1943) arrange behavior into a hierarchy which proceeds from active (as contrasted to passive) to purposeful, to feedback (teleological), and to predictive. Rapoport (1953) carries this model into the area of biological adjustment:

> **Only one direction seems promising—the acquisition of the higher adjustment faculties. This direction can be taken by individuals, groups, and the whole of mankind. It implies a struggle; not a struggle against potential allies, but a struggle against the chaotic tendency of nature, a struggle of creating order out of chaos. . . . (p. 191 f.)**

Hallowell (1959) has discussed this adjustive faculty in terms of ego function. Needham (1943) has related it to social evolution: "Every transition from the unconscious to the conscious implies a step from bondage to freedom, from lower to higher level of organization. All early agriculture and storage of food-products ncessitated more conscious control than before . . ." (p. 264). Huxley (1960:19), too, emphasizes the emergence of awareness and places on man the responsibility for further evolutionary advance—or damage—on this planet. Science, of course, is the most striking of these conscious processes—at least to scientists:

> **With the growth of scientific prediction, man has acquired the greatest capacity for anticipating the future. He can often make the necessary adjustments in advance and control the conditions. . . . There is already some tendency for man to act for the benefit of unborn generations . . . which has a sound basis in the evolutionary dynamics of population systems (Allee *et al.*, 1950: 639 f.).**

The thermodynamic imperative itself may thus be said to have undergone evolution, from the unconscious adaptations of infrahuman organisms, through the development of "exosomatic" culture (Lotka, 1945) by which great stores of information can be transmited to subsequent generations and otherwise "distributed" (Brillouin, 1950), to the scientific descriptions of evolution and the deliberate attempts to increase the availability of energy for survival and the enjoyment of life. Given the insignificance of life on this planet when viewed from astronomical perspectives, these considerations lead to the following ethical statement: While the universe will probably continue to exist and to

change, the likelihood of life-somehow-related-to-life-as-we-know-it surviving for a long time is small, but the chances can be improved by recognition of the thermodynamic imperative and action based upon it.

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STUDIES ON THE CAINGANG INDIANS

I. DEMOGRAPHY

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THE mathematical papers of Fisher, Wright, Haldane and others have greatly advanced our understanding of biological evolution generally. To further our knowledge about the evolutionary forces active in man it was decided to start a long-term study in present-day Brazilian Indian populations. The first stage of this investigation is a study of demographic factors relevant to genetic problems. Genetic data were obtained also concerning color blindness, blood groups, hemoglobin and haptoglobin types, and other characteristics, but their analysis is not included here.

MATERIAL AND METHODS

The subjects under consideration live in nine localities of the State of Rio Grande do Sul, Brazil (figure 1), in State reservations set aside for them. The great majority of these Indians are Caingang, a Gê-speaking tribe whose distribution area extends also to the States of Santa Catarina, Paraná and São Paulo (Métraux, 1946). The rest are small groups of Guaranis. They will not be considered in this paper (for details about them see Salzano, 1960).

The number of excursions to the Indian communities for the collection of the demographic and other genetic information was as follows: Nonoai: six excursions, with a duration of three to four days each; Guarita: four excursions of three to five days each; Ligeiro: three excursions of two days each; Cacique Doble: four excursions of two days each; Inhacorá: one excursion of two days; Água Santa: one excursion of one day; Ventarra: one excursion of one day; Votoro: one excursion of two days; Serrinha: one excursion of one day.

The bulk of the demographic material was obtained in January of 1958 when all these localities were visited for periods ranging from one to three days. Later visits to the larger localities, Nonoai, Guarita, Cacique Doble and Ligeiro, served to complete and expand this information.

The way the data were collected was fairly uniform. On arrival at a locality contacts were made with the State authority responsible for these populations and with the Indian chief designated by the State authority as captain or colonel of police. A meeting of the people to be studied was then arranged.

In the smaller localities only a simple list was made which included name, age, place of birth and racial characterization of the examined persons. This last item was based on visual inspection and sometimes on direct questions about his or her ancestry made to the individual, to the State authority or to members of the Indian police. More detailed data were obtained in the larger localities by filling out a family questionnaire which included the following items: 1. name of the spouses; 2. racial characterization; 3. age; 4. place of birth; 5. consanguinity; 6. number of children: a. alive; b. dead; 7. names and ages of the living children; 8. ages of the dead children; 9. place of birth of all the members of the family; 10. information if someone of the family had left the locality.

The age, especially in elderly people, could be obtained only by estimate. Several questions concerning consanguinity of spouses were asked in order to be sure that the subject had understood, and to determine the exact type of relationship.

RESULTS

Racial characterization of the populations. Table 1 and figure 1 show the number of persons and their race in the nine Caingang Indian communities studied. As can be noticed there is great variability from place to place in the frequency of Indians with no signs of hybridization. In the four larger localities (Guarita, Nonoai, Ligeiro and Cacique Doble) significantly higher frequencies of Indians without signs of hybridization are found—76.5% as compared with 49.8% in the smaller localities ($\chi^2 = 159.23$; 1 d.f.; $P < 0.001$). The variation among the localities, however, within each of these two groupings is great and statistically significant (Larger localities: $\chi^2 = 42.7$; 3 d.f.; $P < 0.001$; Smaller ones: $\chi^2 = 79.8$; 4 d.f.; $P < 0.001$).

Despite efforts to do so, it was difficult to obtain information about all the residents of the localities studied, and there is no doubt that the survey was not complete in most of them. In the two larger groups (Nonoai and Guarita) the census did not include all individuals. In the localities of Ligeiro and Cacique Doble however, the results are

in all probability close to the actual population. In the last column of table 1 the results of the last census made by the State authorities are presented for comparison with the data now collected. It should be pointed out that the official censuses are far from perfect and the results quoted are not to be interpreted as representing the true population of these localities. In general, however, the numbers obtained agree fairly well with the data we obtained.

TABLE 1

Frequencies in per cent of Indians, Mestizos and Neo-Brazilians living in Caingang communities

LOCALITIES	CAINGANG INDIANS	C × B MESTIZOS	GUARANI INDIANS	G × C MESTIZOS	NEO-BRAZILIANS	CORUAIA INDIANS	TOTAL	PREVIOUS INDEPENDENT CENSUS
1—Guarita	76.8	23.0	0.1	—	0.1	—	671	791
2—Nonoai	83.8	13.5	0.2	1.2	1.3	—	605	973
3—Ligeiro	65.7	33.2	—	—	0.7	0.4	271	—
4—Cacique Doble	68.4	31.6	—	—	—	—	193	200
5—Água Santa	41.0	45.3	—	—	13.7	—	95	70
6—Inhacorá	87.5	12.5	—	—	—	—	104	173
7—Serrinha	48.9	50.0	—	—	1.1	—	188	176
8—Votoro	45.7	47.8	—	—	6.5	—	138	268
9—Ventarra	31.8	64.3	—	—	3.9	—	129	168
All localities	69.2	28.6	0.1	0.3	1.7	0.1	2,394	2,819

C × B Mestizos = Caingang/Neo-Brazilian
G × C Mestizos = Guarani/Caingang

Distribution by sex and age. Table 2 presents the distribution by sex and age, of the Caingang Indians and Caingang-Neo-Brazilian Mestizos studied. Notice first the relatively small frequency of young people (less than 15 years of age) among the Indians as compared to the Mestizos, 35.7 and 54.5% respectively ($\chi^2 = 70.9$; 1 d. f.; $P < 0.001$). This seems to indicate a probable rise in the frequency of Mestizos in these localities in future years. When the data concerning Indians and Mestizos are combined, the value for the population aged less than 15 years turns out to be 41.2%, very close to the corresponding value for the general population of Rio Grande do Sul (1950 census), which was 41.4%.

As to the sex-ratio, there is a statistically significant deficiency of men among the Indians, but an excess among the Mestizos.

Inter-racial marriages. Data about the occurrence of inter-racial marriages in the four larger localities are shown in table 3. Mixing is occurring in a different rhythm in the four localities. In Nonoai the majority of marriages are of Indian with Indian (85.9%). This fre-

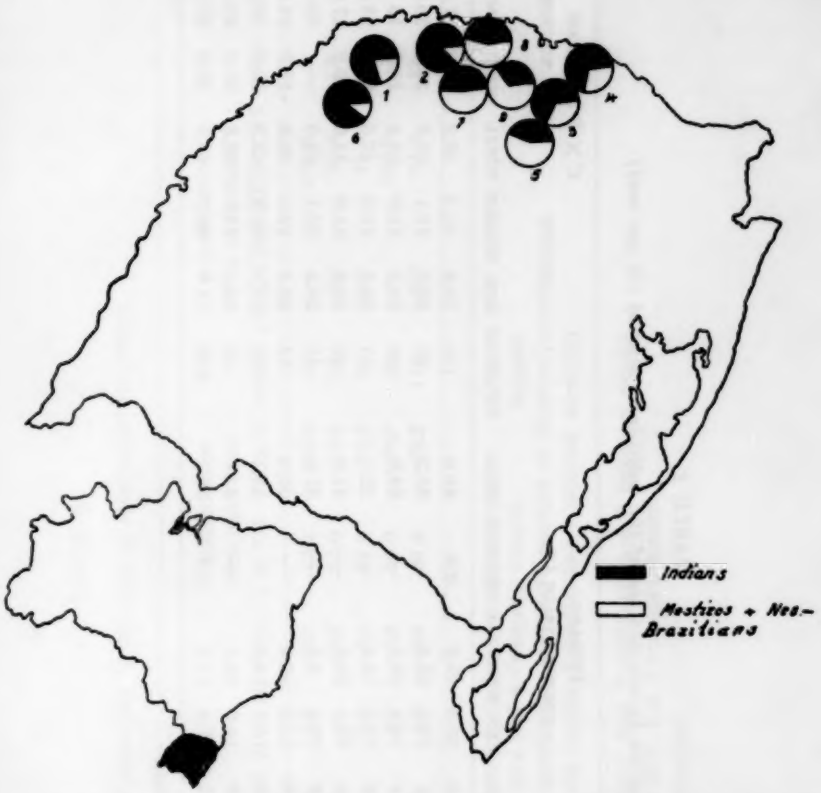

FIG. 1. RACIAL CHARACTERIZATION OF PERSONS STUDIED. SMALL MAP OF BRAZIL (LOWER LEFT) AND ENLARGED MAP OF THE STATE OF RIO GRANDE DO SUL SHOWING THE LOCALITIES STUDIED (NUMBERS CORRESPOND TO THOSE USED IN TABLE 1).

quency, however, diminishes to 50.5%, in the locality of Ligeiro. The differences among these localities are statistically significant ($\chi^2 = 46.3$; 6 d.f.; $P < 0.001$), and parallels the frequencies of Mestizo individuals in these populations. This shows that there has not been a fundamental change between the hybridization rates in the past and that now occurring.

In marriages between Indians and Neo-Brazilians there are ten times

TABLE 2

Discrimination by age and sex of the individuals studied (in per cent)

	Caingang Indians								C × B Mestizos							
		Adults			Younger than 15					Adults			Younger than 15			
Localities	Actual numbers	Men	Women	Total	Men	Women	Unknown	Total	Actual numbers	Men	Women	Total	Men	Women	Unknown	Total
Guarita	515	29.1	30.9	60.0	20.0	19.2	0.8	40.0	154	18.8	23.4	42.2	29.2	27.9	0.7	57.8
Nonoai	507	29.0	32.7	61.7	16.0	22.3	—	38.3	82	20.7	17.1	37.8	36.6	25.6	—	62.2
Ligeiro	178	30.3	40.5	70.8	14.0	15.2	—	29.2	90	35.5	18.9	54.4	28.9	16.7	—	45.6
Cacique Doble	132	34.9	42.4	77.3	12.1	10.6	—	22.7	61	26.2	21.3	47.5	29.5	23.0	—	52.5
Água Santa	39	28.2	30.8	59.0	15.4	25.6	—	41.0	43	20.9	21.0	41.9	30.2	27.9	—	58.1
Inhacorá	91	34.0	28.6	62.6	28.6	8.8	—	37.4	13	38.4	23.1	61.5	—	38.5	—	38.5
Serrinha	92	39.1	30.5	69.6	15.2	15.2	—	30.4	94	23.4	19.1	42.5	33.0	24.5	—	57.5
Votoro	63	34.9	36.5	71.4	14.3	14.3	—	28.6	66	30.3	18.2	48.5	18.2	33.3	—	51.5
Ventarra	41	36.6	29.3	65.9	14.6	19.5	—	34.1	83	24.1	24.1	48.2	25.3	26.5	—	51.8
All localities	1,658	30.9	33.4	64.3	17.3	18.2	0.2	35.7	686	24.8	20.7	45.5	28.6	25.8	0.1	54.5

C × B Mestizos = Caingang/Neo-Brazilian

TABLE 3

Frequencies in per cent of inter-racial marriages

LOCALITIES	I × I	I × M	I × NB	M × NB	M × M	TOTAL	DIRECTION OF THE GENE FLOW [c] ♂I × ♀M	♂M × ♀I	TOTAL	DIRECTION OF THE GENE FLOW [c] ♂I × ♀NB	♂NB × ♀I	TOTAL	CHILDLESS INTER-RACIAL MARRIAGES	TOTAL
Guarita	73.8 [a]	19.1	—	0.5	6.6	183	43.2	56.8	44	—	100.0	5	16.7 [d]	48
Nonoai	85.9 [b]	7.4	2.4	4.3	—	163	25.0	75.0	12	7.7	92.3	13	8.7 [e]	23
Ligeiro	50.5	39.6	3.3	—	6.6	91	27.8	72.2	36	25.0	75.0	4	40.0 [f]	45
Cacique Doble	60.3	32.8	—	—	6.9	58	50.0	50.0	20	—	100.0	1	34.8 [g]	23
All localities	71.9	20.6	1.4	1.6	4.5	495	37.5	62.5	112	8.7	91.3	23	25.9	139

[a] One of the marriages was between a Guarani man and Caingang woman.
[b] Two marriages were between a Guarani man and a Caingang woman.
[c] Part of the data obtained also through questioning about the parents of Mestizo persons.
[d] Two unknown causes, 3 children died, 1 old couple, 1 sterile, and 1 married recently.
[e] One sterile, and 1 child died.
[f] Seven married recently, 2 sterile, 6 children died, 1 stillborn, and 2 unknown causes.
[g] Three married recently, 3 sterile, 1 child died, and 1 unknown causes.
I = Indian; M = Mestizo; NB = Neo-Brazilian.

as many marriages between Neo-Brazilian men and Indian women as the reverse. There is also a marked asymmetry in the marriages between Indians and Mestizos with an excess of marriages between Mestizo men and Indian women. This situation is due, in part, to the differences in the sex-ratio between the two groups. There is a deficiency of Indian and excess of Mestizo men (both adult and young). The differences in sex-ratio do not completely explain the facts, for the frequency of marriages between Indian men and Mestizo women is lower than would be expected if the persons chose their spouses at random in respect to race ($\chi^2 = 4.5$; 1 d.f.; $P < 0.05$).

TABLE 4

Frequencies in per cent of endogamous and exogamous marriages in the localities under study

LOCALITIES	BOTH PARTNERS FROM SAME LOCALITY	ONE PARTNER FROM ANOTHER LOCALITY	BOTH FROM OUTSIDE	TOTAL	MAIN SOURCE OF IMMIGRATION	NUMBER OF IMMIGRANTS	NUMBER OF LOCALITIES WHICH GAVE IMMIGRANTS
Guarita	89.2	10.2	0.6	167	Inhacorá (21.4)	14	11
Nonoai	60.9	31.4	7.7	156	Serrinha (37.8)	74	15
Ligeiro	60.0	30.0	10.0	90	Cac. Doble (58.7)	46	10
Cacique Doble	86.0	10.5	3.5	57	Ligeiro (81.8)	11	3
All localities	73.8	21.1	5.1	470	—	—	—

Of course inter-racial marriages have genetic implications only when the union is fertile. Table 3 presents data about childless inter-racial marriages in the localities studied. These frequencies do not differ significantly from those in intraracial marriages (derived from table 7). There is variability, however, in the frequency of childless inter-racial marriages in the different localities studied ($\chi^2 = 11.3$; 3 d.f.; $P < 0.02$), but this parallels that in all marriages (table 7), and within each locality there was no significant difference between the intra and inter-racial marriages.

Isolation. The frequency of marriages in which both spouses are from the same locality or region varies with the mobility of the population and the degree of geographical isolation. In the populations under study the frequency of these marriages, as shown in table 4, varied

between 60.0% to 89.2%. The variation is statistically significant ($\chi^2 = 49.3$; 6 d.f.; $P < 0.001$).

As can be seen from the last three columns of table 4 58.7% of the Ligeiro immigrants came from Cacique Doble while in the latter locality the percentage of immigrants from Ligeiro is still higher: 81.8%. These two small communities are geographically very close and are similar in

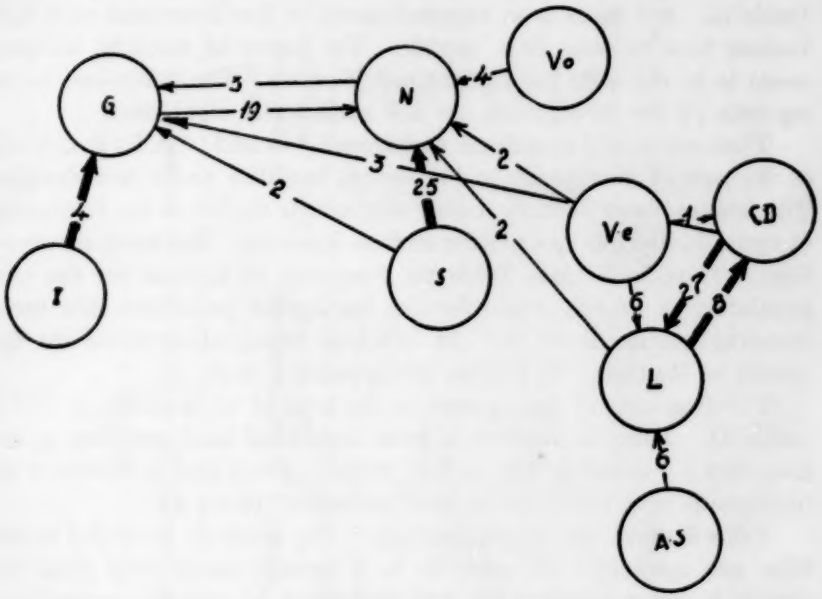

FIG. 2. BIRTHPLACES OF PERSONS BORN IN INDIAN LOCALITIES OF THE STATE WHO MARRIED IN GUARITA (G), NONOAI (N), LIGEIRO (L), AND CACIQUE DOBLE (CD). I = INHACORÁ; S = SERRINHA; VO = VOTORO; VE = VENTARRA; AS = ÁGUA SANTA. ARROWS INDICATE THE DIRECTION OF MIGRATION; THE WIDER ARROWS CORRESPOND TO THE MAIN SOURCES OF SPOUSES BORN OUTSIDE THE LOCALITY.

several respects. Nonoai receives immigrants from more sources than does Cacique Doble for instance, since immigrants from 11 localities had established themselves in the former while immigrants from only three different localities occur in the latter.

Figure 2 shows additional data concerning this point. It indicates birthplaces of the persons born in the Indian localities of the State who married in the four communities under study. The figure indicates the

importance of the geographic distance factor in these unions, showing two blocks of localities, a group gravitating in function of Guarita and Nonoai; and the other in function of Cacique Doble and Ligeiro.

Immigration and emigration. Does race influence the migration of individuals in the populations under study? As can be seen in table 5 there are no great differences between the frequencies of Indians and Mestizos in the migrant population when compared to the total population (table 1). But there is an expected excess of Neo-Brazilians since non-Indians have to come from outside. The degree of mobility, however, seems to be the same for Indians and Mestizos. The differences in the sex-ratio of the immigrants are not statistically significant.

There are locally significant variations ($\chi^2 = 44.5$; 6 d. f.; $P < 0.001$) in the race of immigrants to the several localities under consideration. The frequencies of Mestizos among migrants is similar to the frequencies observed for the total population of these localities. But there are exceptions. Thus, in Cacique Doble the frequency of Indians for the total population is 68.4%, while for the immigrant population the corresponding number is 45.5%. In this case immigration raised the frequency of Mestizos. In Ligeiro the opposite is true.

The frequency of immigrants in the total of all localities is 11.2% (table 5). There is, however, a great amount of local variation in this frequency ($\chi^2 = 282.3$; 6 d. f.; $P < 0.001$). From 60.9 to 92.9% of the immigrants contributed to the next generation (table 5).

Table 6 shows the emigration data. The numbers presented in this table are not large, but coincide to a certain extent with what was already known concerning the gene flow which is occurring among these populations. Thus at Guarita only 1.2% of the population are known to have emigrated to Nonoai (probably the number is a little higher than this, but not much) while the community received the equivalent of 2.1% of its population as immigrants (table 5). Nonoai receives a much higher percentage of immigrants (12.2%) than its supply of emigrants (2.0%). And Ligeiro and Cacique Doble once more show the genetic exchange existing between these localities since, as shown before, the main source of absorption of emigrants of Ligeiro is Cacique Doble and vice versa (see also figure 2).

Family sizes. As can be seen in table 7 in these localities the average number of children per couple is 2.2 ± 0.1, which is very low. Without details of marriage duration or age it is not possible to say how far these populations are from the replacement level, for which a rough

TABLE 5

Racial characterization of the immigrants (in per cent)

LOCALITIES	INDIANS			MESTIZOS			NEO-BRAZILIANS			TOTAL	% OF THE TOTAL POPULATION	IMMIGRANTS WHO CONTRIBUTED TO THE NEXT GENERATION (%)
	M	W	T	M	W	T	M	W	T			
Guarita	21.4	35.7	57.1	21.4	21.5	42.9	—	—	—	14	2.1	92.9
Nonoai	47.3[a]	39.2	86.5	2.7	2.7	5.4	5.4	2.7	8.1	74	12.2	62.2
Ligeiro	30.4[b]	47.8	78.2	10.9	6.5	17.4	2.2	2.2	4.4	46	16.9	60.9
Cacique Doble	18.2	27.3	45.5	9.1	45.4	54.5	—	—	—	11	5.7	63.6
Serrinha	9.1	45.4	54.5	18.2	18.2	36.4	9.1	—	9.1	11	5.8	—
Votoro	25.0	23.5	48.5	26.5	14.7	41.2	5.9	4.4	10.3	68	49.3	—
Ventarra	9.5	14.3	23.8	47.6	14.3	61.9	4.8	9.5	14.3	21	16.3	—
All localities	30.2	33.9	64.1	16.7	11.4	28.1	4.5	3.3	7.8	245	11.2	64.8

[a] 2 Guaranis. [b] 1 Coruaia. M = Men; W = Women; T = Total

minimal estimate from table 8 is 3.4. The populations of Guarita and Nonoai seem to be farther from this level than those of Cacique Doble and Ligeiro.

Mortality before the age of reproduction. The mortality rates before the age of reproduction are very high in the localities under study as can be seen in table 8. The number for all localities is 41.0%. No statistically significant differences in the mortality rates could be found, in the several localities studied.

TABLE 6

Emigration *

LOCALITIES	NO. OF EMIGRANTS	% OF THE TOTAL POPULATION	MAIN SOURCE OF ABSORPTION	BIRTHPLACE IN THE ISOLATE	BIRTHPLACE OUT	CONTRIBUTION TO THE LOCAL GENE POOL +	CONTRIBUTION TO THE LOCAL GENE POOL —
Guarita	8	1.2	Nonoai	6	2	—	8
Nonoai	12	2.0	Votoro	8	4	1	11
Ligeiro	5	1.8	Cacique D.	5	—	—	5
Cacique Doble	5	2.6	Ligeiro	4	1	1	4
All localities	30	1.7	———	23	7	2	28

* Due to the way the data were collected, no numbers could be obtained regarding the migration of whole families.

Childless marriages. Table 9 shows the frequency of childless couples married more than two years. The results were separated by generations. The length of one generation was estimated to be 21 years for the populations under consideration. The estimate for Brazil in general (Mortara, personal communication) is 30 years. But marriages occur much earlier among the Indians than in other Brazilians (almost all Indians are married at the age of 15). Therefore the age of 21 years seemed to be more indicated for this study.

In all localities the percentage of childless marriages is 5.2, indeed very high. But there is great heterogeneity in the populations studied. Nonoai has only 2.7% of childless couples, Cacique Doble 14.6%. The differences among localities are statistically significant ($\chi^2 = 11.0$; 3 d. f.; $P < 0.02$). It is unlikely that these differences are due to age differences

TABLE 7

Family sizes (in per cent)

NO. OF CHILDREN / LOCALITIES	0	1	2	3	4	5	6	7	8	14	NO. OF FAMILIES	MEAN NUMBER OF CHILDREN
Guarita	24.3	20.0	15.7	14.6	5.4	5.9	8.1	3.8	2.2	—	185	2.4 ± 0.2
Nonoai	19.1	19.8	18.5	15.4	13.6	4.9	5.6	3.1	—	—	162	2.4 ± 0.1
Ligeiro	30.0	36.7	10.0	7.8	7.8	4.4	1.1	2.2	—	—	90	1.6 ± 0.2
Cacique Doble	33.3	21.0	22.8	12.3	1.8	3.5	3.5	—	—	1.8	57	1.7 ± 0.3
All localities	24.7	23.1	16.4	13.4	8.1	5.0	5.5	2.8	0.8	0.2	494	2.2 ± 0.1

TABLE 8

Mortality before the age of reproduction

LOCALITIES	NUMBER OF BIRTHS	DEATHS BEFORE THE AGE OF 15 (%)
Guarita	768	41.0
Nonoai	681	41.7
Ligeiro	262	43.9
Cacique Doble	156	32.7
All localities	1,867	41.0

TABLE 9

Childless marriages by generation

	GENERATIONS							
LOCALITIES	3rd	2nd	1st	2nd3rd	1st2nd	1st3rd	AGE OF ONE PARTNER UNKNOWN	TOTAL
Guarita								
marriages	80	36	20	11	12	1	2	162
childless	7(8.7)	0	0	0	0	0	0	7(4.3)
Nonoai								
marriages	65	32	22	19	9	3	1	151
childless	1(1.5)	1(3.1)	2(9.1)	0	0	0	0	4(2.7)
Ligeiro								
marriages	32	16	11	14	3	—	1	77
childless	1(3.1)	1(6.3)	2(18.2)	1(7.1)	0	—	0	5(6.5)
Cacique Doble								
marriages	26	13	4	5	—	—	—	48
childless	6(23.1)	1(7.7)	0	0	—	—	—	7(14.6)
All localities								
marriages	203	97	57	49	24	4	4	438
childless	15(7.4)	3(3.1)	4(7.0)	1(2.0)	0	0	0	23(5.2)

among the couples living in the four localities, for the latter are not statistically significant, though the percentage of childless couples in the third (youngest) generation is much higher in Guarita and Cacique Doble than in Nonoai and Ligeiro.

Consanguinity. For the knowledge of the genetic structure of a population it is necessary to calculate its inbreeding coefficient, which is obtained by establishing the frequency rate of consanguineous marriages. As can be seen in table 10, the total frequency of such marriages in

TABLE 10

Frequencies in per cent of consanguineous marriages in the localities under study

LOCALITIES / TYPE	GUARITA	NONOAI	LIGEIRO	CACIQUE DOBLE	ALL LOCALITIES
Uncle/niece	0.6	0.7	1.2	—	0.6
1st cousins	8.1	3.3	1.1	1.9	4.5
1st cousins once removed	5.2	0.6	2.3	—	2.6
2nd cousins	7.5	2.0	6.9	5.6	5.4
2nd cousins once removed	0.6	—	—	—	0.2
Double second cousins	0.6	—	—	—	0.2
Others	0.5	—	—	1.9	0.5
Total frequency	23.1	6.6	11.5	9.4	14.0
Total number of marriages	173	152	87	53	465
Inbreeding coefficient	0.009	0.003	0.004	0.002	

the four populations under study is high: 14.0% as compared with a frequency of 2.4% estimated by Freire-Maia (1957) for the State of Rio Grande do Sul (white populations). There is significant local variation in the frequency with which these types of unions occur in the Indian communities ($\chi^2 = 20.3$; 3 d.f.; $P < 0.001$).

The inbreeding coefficients omitting the two marriages in which the degree of consanguinity was not established with certainty, indicated as "others" in table 10, varied between 0.009 and 0.002.

Effective population sizes. In a natural population the total number of individuals of all ages may be very large, but not every individual reaches sexual maturity and mates. Those who mate do not necessarily leave offspring who survive to maturity in the next generation. The number of actual progenitors (breeding size) responsible for the genetic constitution of the next generation may be much less than the number of individuals living at any time in a population. Further the breeding size may be much larger than the conceptual "effective" size of a population, an "ideal" population where there are N breeding individuals, half of them females and half males mating at random, and where the variance of the random deviation of gene frequencies is $q(1-q)/2N$ and the rate of decay of variability $1/2N$.

Gene frequencies are subject to random fluctuations from one generation to another, quite independently from the changes induced by mutation, selection and migration. Effective size is a useful tool by which effects of these changes can be quantified in any given population.

One formula (Wright, 1938, 1940) for the effective size of a population which has been applied to a number of human populations since Lasker's (1952) study is

$$Ne = \frac{4N-2}{\sigma k^2+2}$$

where N is the number of parents in the population and σk^2 is the variance in the number of gametes contributed to the next generation.

This formula is not strictly applicable to the present data since these populations are not numerically stable but for interest the values are shown in table 11. Such numbers represent from 37.7 to 41.7% of the total populations of these communities. The effective sizes found were: 42, 91, 145 and 176. These figures are underestimates, *e.g.* by the extent to which the present count was incomplete though perhaps its percentage (from 21.6 to 33.6%) of the total populations is a reasonable approximation.

Glass *et al.* (1952) suggested that in the Dunker isolate of Pennsylvania the best estimate of the effective size would be the simple enumeration of parents in one of the present generations. In the results here obtained generation length being taken as 21 years, if the number of persons of the third generation who contributed to the next generation is taken as representative of the effective sizes of these populations (thus avoiding mortality effects), it can be seen in table 11 that they would be

43, 59, 123 and 136, the percentages in relation to the total population varying between 18.3 and 22.5%.

Random drift in a population per generation in the absence of

TABLE 11

Effective population size and admixture rate in Caingang communities

	Guarita		Nonoai		Ligeiro		Cacique Doble	
	No. indiv.	% of total pop.	No. indiv.	% of total pop.	No. indiv.	% of total pop.	No. indiv.	% of total pop.
1st generation	46	6.8	38	6.3	17	6.3	8	4.1
2nd generation	84	12.5	73	12.1	36	13.3	25	12.9
3rd generation	123	18.3	136	22.5	59	21.8	43	22.3
Unknown	—	—	2	0.3	1	0.4	—	—
Breeding size	253	37.7	249	41.2	113	41.7	76	39.4
Total population	671	—	605	—	271	—	193	—
Effective size	145	21.6	176	29.1	91	33.6	42	21.8
Admixture rate (m)	5.7	—	23.4	—	25.0	—	8.8	—
$\sigma^2 dq = \frac{q(1-q)}{2Ne}$ ($q = 0.50$)	0.001	—	0.001	—	0.001	—	0.003	—
Nem	8	—	41	—	23	—	4	—

immigration, selection and mutation pressures may be calculated as follows (Wright, 1940):

$$\sigma^2 dq = \frac{q(1-q)}{2Ne}$$

where $\sigma^2 dq$ is the variance due to drift, q represents the gene frequency and Ne the effective population size. The highest deviations occur when q and $1-q$ are 0.5 (Li, 1955; Glass, 1954).

$\sigma^2 dq$ calculations for the populations studied are presented in table 11 for the particular case of gene frequencies of 0.5. The values obtained varied between 0.001 and 0.003.

Wright (1940, 1943) has shown that differentiation due to drift for a given gene frequency in the absence of selection and mutation pressures depends upon the product of the effective population size and the migra-

tion rate (Nem, where m is the rate of migration). For the calculation of m, the admixture rate (Lasker, 1954), the proportion of parents is used. Table 11 shows the values for Nem obtained; in Cacique Doble, where the value is 4, drift likely will have led to important differentiation if the conditions have persisted for a number of generations.

DISCUSSION

Appraising the significance of the findings reported above, from the factors which influence hybridization in these populations, one could conclude that in the larger localities, where there is more probability of marriage within the race, the frequency of Mestizos should be lower. This was observed in some cases. For instance Nonoai and Guarita, the largest localities, showed high frequencies of Indians without signs of hybridization. But population size probably is not the only factor involved. Inhacorá, for instance, is a small locality and has a low percentage of hybrids (table 1). The isolation of the Nonoai Indians from the Neo-Brazilian populations is more extreme than in the Ligeiro reservation and this of course influences the degree of mixture. Cultural factors play a part, too, producing different attitudes toward hybridization.

The higher frequency of marriages between Neo-Brazilian men and Indian women, against the converse type of union, has a very simple explanation. Many Neo-Brazilian men try to marry Indian women in order to obtain land in the reservations without payment to start their plantations. The asymmetry present in the marriages of Indians and Mestizos is more difficult to explain since their social status is identical. It seems, however, that Indian men prefer to marry Indian women. Because these women are in excess many Mestizo men marry them also, which is natural since there are fewer Mestizo women than Mestizo men in these communities. The fertility seems to be the same in intra and inter-racial marriages.

As to the age structure of these populations it was pointed out already that the frequency of individuals aged less than 15 years is quite close to the corresponding frequency in the general population of the State (41.2% against 41.4%, respectively). In populations culturally more primitive this frequency is generally more elevated. For instance Roberts (1956) found 47.7% of persons aged less than 15 years among the Dinkas of Sudan.

The degree of local marriage in the Indian communities of Rio Grande do Sul is very high, indeed it is higher than the frequency of

marriages within a community reported by Sutter (1958) in France, almost one hundred years ago! The numbers obtained in the present study are similar, however, to those obtained by Lasker (1954) in the Mexican community of Paracho. Geographical isolation seems to have established two different evolutionary units, regarding the four localities studied. One formed by Cacique Doble-Ligeiro and the other by Nonoai-Guarita (figure 2). This impression, obtained through the analysis of demographic data, was fully confirmed by the genetic data to be reported elsewhere.

Geographic distance is not the only factor which regulates the gene flow among these communities. This becomes clear once one realizes that there was much more interchange between Serrinha and Nonoai than between this last locality and Votoro, despite the fact that the distances which separate Nonoai from these two other localities are approximately the same. The probable explanation is that the Passo Fundo river prevents a closer contact between the populations of Votoro and Nonoai. The construction of a bridge which is being finished now and of a good highway connecting the two regions will probably change this situation in the near future.

The demographic instability of the majority of the South American Indian populations has preoccupied many investigators. Despite some optimistic claims (*e. g.*, Rosenblat, 1945), it is generally held that these populations have suffered a large reduction in numbers since the epoch of European contact. For the Brazilian Indians this reduction has been estimated from 55 (Steward, 1949) to 90% (Ribeiro, 1957). What is the situation in regard to the Indian population of Rio Grande do Sul? Data gathered by Laytano (1955, 1956, 1957a and b) and reviewed elsewhere (Salzano, 1960), seem to indicate that the Indians from Rio Grande do Sul, already integrated in the Brazilian life (Ribeiro, 1957), have reached a state of relative demographic equilibrium and may even increase in number in future years.

But the situation, in the communities studied in this paper, is different. None of these localities is at the replacement level and consequently they will decrease in numbers in future years unless the present situation is changed. This is due to the high mortality before the age of reproduction and the high frequency of childless marriages. 41% of the persons born in these localities will not reach the age of reproduction. Data about children's survival for Brazil's general population gives a much lower figure, 25.0% for all ages (Mortara, 1957). Information

about this problem in primitive populations is very limited, most of it being based on indirect evidence but generally the mortality is of the same order of magnitude as those reported here.

The differences in family size in the four localities studied are in great part due to different frequencies of childless marriages, since mortality before the age of reproduction is about the same in the four groups. The higher percentages of childless marriages in the third (youngest) generation in Guarita and Cacique Doble probably indicates that at least in these localities, artificial control of natality is being performed.

The variability found in the inbreeding coefficient of the populations studied is difficult to explain. It does not parallel the degree of endogamy. It could be expected that smaller populations would have a higher frequency of consanguineous marriages, due to the impossibility of their inhabitants choosing unrelated partners, but this relation was not found. The two larger localities (Guarita and Nonoai) represented the extremes in the variation of this frequency. It is possible that cultural factors have influenced the behavior of these groups in relation to this problem between communities and there may of course be differences in the amount of inbreeding that the method of investigation failed to reveal.

The data obtained about effective size in relation to the total population size agree with those obtained by other authors. The estimates gave a percentage between 21.6 and 33.6 (table 11), while those calculated by Glass *et al.* (1952) and Lasker (1954) among the Dunkers and in Paracho yield 25.7 and 21.0%, respectively, of the total populations.

Calculations from effective population sizes, indicate that with the present demographic structure in Cacique Doble important differentiation may be occurring due to genetic drift in relation to some genes though this is not to say that it was formerly equally important. In the other localities the differentiation due to this factor may not be very important, but it cannot be overlooked. The genetic data collected, which we hope to report in the near future, present some results which can be explained only by postulating the action of this factor at least in Cacique Doble.

SUMMARY

There exists local variation in the amount of gene flow between the Indian and neighboring populations. The Mestizo populations are generally younger than the Indian. Differences in sex-ratio were found between the Indian and Mestizo populations. All the populations present

some degree of isolation. Distance has produced two separate evolutive complexes. But other factors influence the quantity of local mating which exists. The racial condition does not influence the mobility of the individuals in these populations. In none of these localities is a replacement level attained. The low number of children per family is due to the high mortality before the age of reproduction and the high frequency of childless couples. The inbreeding coefficients are fairly high. The effective sizes of these populations constitute from 21.6 to 33.6% of the total populations. Genetic drift is perhaps effective at least in one locality in determining differentiation in some loci.

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THE ROLE OF IMMUNOCHEMICAL DIFFERENCES IN THE PHYLETIC DEVELOPMENT OF HUMAN BEHAVIOR[1]

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THE phyletic development of human behavior involved factors which operated at such different levels as the biochemical, the anatomical, the ecological, and the psycho-sociological. Our concern will be with the immunochemical level and its dynamic interactions with the other levels.

A key factor at a critical stage of the process was the arboreal mode of life of the ancient Primates. As a consequence of this mode of life natural selection early in the history of the Primates set in motion an expansion of the brain (Le Gros Clark, 1959). The anatomical specializations characterizing other orders of mammals were avoided. Primitive features such as pentadactyl limbs were preserved, and the nervous system which was developed permitted such adaptive features as steroscopic vision, dexterity of fingers, and superb coordination between hand and eye. Eventually the way was paved on the basis of intelligence (as well as strength due to increasing size) for the most progressively evolving Primates to contend successfully with numerous predators on the ground. By the time of the Pleistocene, bipedal hominids were employing implements as part of their mode of existence. The use of tools and weapons associated with carnivorous hunting habits then became the decisive element in the emergence of Homo sapiens (Washburn and Avis, 1958). There were now new opportunities for the selection of intelligence, and when man appeared, he was in some respects a new type of being, one with a remarkably elaborated brain, an unrivaled power of speech, and portentous possibilities for cultural evolution.

[1] Presented in part at a conference on biochemical anthropology, July 6-8, 1960, at the Fels Research Institute, Yellow Springs, Ohio. The conference, organized by Dr. Stanley M. Garn, was sponsored by the Wenner-Gren Foundation for Anthropological Research. This study has been supported by grant M-2476 of the National Institute of Mental Health, grant 226 of the National Multiple Sclerosis Society, and grant G14152 of the National Science Foundation.

The roots of human behavior can also be traced to the immaturity of the new born mammal, to the dependency of the mammalian offspring on the mother as exemplified by the nursing habit. In this connection, Pavlovian researches have established that in animals immature at birth the slower development of the central nervous system creates the circumstances "for the more prolonged action of factors in the surroundings on the developing mechanism for inborn reflexes, and this leads to considerable reshaping of hereditary forms of behavior and the development of more perfected forms of conditioned behavior" (Volokhov, 1959, p. 51). In other words, the protracted immaturity of the mammalian offspring favors the development of learned behavior.

It should be emphasized this is a special kind of immaturity, one based first on the intimate placental connection of fetus to mother which provides for effective and prolonged prenatal nourishment, then on the crucial period after birth during which the infant still depends on the mother for nourishment and protection. Only those mammalian populations with an advancing level of psychological reactivity could have endured the evolutionary process which caused a lengthening of the period of postnatal dependency. In turn, as a consequence of a delayed maturity, new opportunities were created in the offspring for versatile and intelligent behavior. The Primates, more so than other mammals, gave full expression in their phylogenesis to the dynamics of the process. As intelligence advanced in the Order, organisms evolved with markedly protracted juvenile phases.

In the following sections, we shall define the genetic nature of this evolutionary process and develop the argument that immunochemical differences between individuals provide a powerful force driving the process. In effect, we shall explore more fully certain of the implications of a previously presented hypothesis (Goodman, 1960a) concerning the effects of protein antigenicity on the evolution of human beings.

GENETIC VARIABILITY AND PROTEIN STRUCTURE

The evolution of slowly maturing organisms results when there is preferential selection for genotypes in which a number of the genes show a delay during ontogeny in expressing their information in protein synthesis. Such selection can occur more readily in a species which has, in the sense of population genetics, a large store of genetic variability, in other words, to follow Fisher's (1930) analysis of genetic variability, a numerous species in the main stream of evolution. If this species

continues in the main stream of evolution, the variability of the ontogenetically-delayed-acting genes will be maintained as the variability of the early acting genes decreases.

These postulated genetic changes may be analyzed in terms of the effects of genic mutations on proteins. If (after Crick, 1958, and others) we consider a gene a coded sequence of desoxyribonucleotides whose informational content is directly reflected by the sequence of amino acids in a polypeptide chain, a genic mutation (in other words, a change in desoxyribonucleotide sequence) must then alter the amino acid sequence of a specific polypeptide. However, the fate of the mutant gene will depend on how the changed amino acid sequence affects the resultant secondary and tertiary structure, the three-dimensional architecture assumed by the polypeptide chains as they fold into integral molecules, since the biological activity of the protein is manifested at this final "phenotypic" level of structure.[2] In determining whether a mutation affecting a particular protein is favorable, detrimental, or neutral to the survival of an organism, we have to consider not only the effect of the mutation on the tertiary or "phenotypic" structure of the protein, but must also consider its effects, if any, on the time and place during ontogeny at which synthesis of the protein begins, on the rate of synthesis of the protein finally achieved, and on the fluctuations of this rate under varying environmental stimuli. However, these effects of time, place, and concentration are very much related to the enactment of the physiological role of the protein. Thus our inquiry can rightly start with the effects of genic mutations on the tertiary or "phenotypic" structure of proteins.

The physiological functioning of a protein at the appropriate places within an organism depends on the nature of the surface-reactive chemical configurations exposed at the level of tertiary structure. Schematically, these can be subdivided into two major categories, the first of which concerns the active centers involved in the primary biochemical reactivity of the protein. We know from the structural analyses of enzymes and hormones that relatively small areas of the total surface of the integral protein molecules constitute the active centers (*e.g.*, Li, 1957; Smith, 1957); also from studies of species specificity (*e.g.*, Tristram, 1953;

[2] The structural levels of proteins are discussed in a large number of publications. A few illustrative references may be cited: Linderstrøm-Lang (1952); Tristram (1953); Porter (1953); Boyer and Schulz (1959); Anfinsen (1959); Scheraga (1959); Perlman and Diringer (1960).

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Porter, 1953), that in evolution a protein type will undergo considerable variation in amino acid composition and antigenic structure without losing its specific, primary physiological, activity. To have utility, the proteins of all organisms, whether primitive or advanced, must exhibit this first category of surface configurations. The second category concerns those ancillary configurations which permit a protein to cross preferentially certain membrane barriers and to be taken up preferentially by certain cell types and not others.

An example of how the tertiary structure of a protein is involved in functions ancillary to the one executed by the active center is provided in a number of mammals by the transport of gamma globulin from the maternal blood stream to the fetal blood stream. Bangham and Tee (1958) found in the rhesus monkey that gamma globulin, apparently in preference to other serum proteins, goes through the placenta into the fetus in especially large amounts. (This preferential transport of antibody protein can be looked upon as an evolutionary adaptation for the neonatal organism, protecting it from infections until its own immunological system matures.) In this connection, Brambell (1958) has demonstrated that the homologous gamma globulin of a mammalian species, upon administration to a pregnant female, can cross into the fetus more readily than gamma globulin from a heterologous animal source. Brambell took these results as indicative of the high specificity of the structural groupings permitting such selective transport. These examples demonstrate that the second category of surface configurations assumes a growing importance for the action of natural selection in the phyletic development of higher organisms.

In our primordial ancestors, then, in whom the physiologically significant effects of the tertiary or "phenotypic" structure of a protein were largely limited to the first category of surface configurations, a large number of mutant genes could accumulate in the population without encountering selective pressures. It may be worth noting that the disulfide bond which contributes significantly to tertiary structure (*e.g.*, Perlmann and Diringer, 1960; Boyer and Schulz, 1959) would not have been present in the primordial proteins of the organisms which lived when a reducing atmosphere still covered the earth. Such proteins, then, must have had much less tertiary structure than modern proteins. For this reason alone, only the active centers of the primordial proteins were likely to have had significance for natural selection, since (to take an extreme position) additional stable surface configurations which

potentially could acquire functions ancillary to the active centers may not even have been present in these proteins.

Apparently, our unicellular, undifferentiated ancestors, as a result of their great store of genetic diversity, were preadapted to give rise to higher forms of animal life, once the environmental conditions had materialized. This appears to have happened when the atmosphere of the earth was changed by photosynthesizing organisms from a reducing one to an oxidizing one (about 800 to 700 million years ago, according to Oparin, 1957). Only then did it become possible for natural selection to perfect the enzymatic machinery for aerobic respiration. Presumably the ingredients for the initial perfection of this machinery were already present in the great store of previously neutral allelomorphic genes. All that was needed was molecular oxygen to act as the selective agency to bring these genes into play and cause their spread through successive populations.

The absence of a significant Precambrian fossil record for metazoan animals and the "sudden" appearance of fossils for many of the major phyla in the early Cambrian (starting about 600 to 500 million years ago) continues to be somewhat of a mystery (Simpson, 1949 and 1960). The treatment above offers an explanation; firstly, the evolution to more complex forms of animal life would occur only after the atmosphere changed from a reducing to an oxidizing one. Then the potentialities of the tremendous store of genetic variability in these primordial forms due to a vast accumulation of previously neutral mutations would find expression. Then, no longer neutral, various different combinations of genes would be sorted out in diverging populations, which in turn would cause the parallel (in contrast to sequential) rise of many of the major animal phyla.

Aerobic respiration, of course, releases much larger quantities of energy than the anaerobic degradative processes. Thus, the organisms with a perfected machinery for aerobic respiration had met at least one requirement for synthesizing the variety of specialized, subtly structured proteins needed by the higher metazoans with their differentiated tissues and homeostatic mechanisms. The ensuing evolution of organisms which harnessed this new source of energy has required, at each stage of phylogenetic advance, a more integrated genotype whose informational content would be differentially expressed throughout ontogeny in the synthesis of various types of proteins. In this way, each phylogenetic advance lessens the probability that the effect of a mutant gene on protein synthesis is neutral to natural selection.

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To recapitulate, mutations now have a better chance of altering physiologically significant aspects of the tertiary structure of proteins and also of altering the epigenetic histories of the proteins. We know from genetic-evolutionary theory that most such alterations (particularly those affecting embryological development) will be detrimental and that the genes which caused them will have a very low frequency in the population. However, some of the alterations, (and these are likely to appear first in the later stages of ontogenetic development), will provide for new beneficial adaptations to the environment. Also, as we know from examples of balanced polymorphisms, certain heterozygous combinations of genes will be particularly beneficial. Thus, even at the phylogenetic level of the birds, the store of genetic variability held by a major species will be formidable, and part of this genetic variability will be due to a still extensive accumulation of relatively neutral mutations.[3]

IMMUNOCHEMICAL DIFFERENCES AND PROGRESSIVE ADAPTABILITY

With the rise of the mammals, a powerful new selective agency acts to limit the population variability of the genes which become active in protein synthesis during the early ontogenetic stages of development. This new selective agency is the capacity of the mother to respond immunologically to antigens of the fetus. Now, practically none of the genic mutations which alter the tertiary structure of proteins can be considered neutral, for any such alteration in tertiary structure, if consummated in the prenatal organism, is likely to give rise to configurations which are potentially antigenic to the mother. A mutation of an early ontogenetically acting gene in a mammal will, therefore, more probably be detrimental to its bearer than one in a bird.

The RH and ABO incompatibilities illustrate this particular danger of a maternal immunological attack on the fetus. The full magnitude of this type of threat is now becoming apparent as a result of the recent intensive study of the mechanisms of transplantation immunity (*e.g.*, Medawar, 1959; Billingham, 1959). The fetus, in a very real sense, simulates a homograft. In this connection, Thomas (1959) has offered

[3] This treatment varies from the approach of Fisher (1930) that denies the possibility of any sizeable number of neutral mutations accumulating. In defense of the position presented here, it may be mentioned that the Fisher approach does not take cognizance of the relevant information concerning protein structure that forms the basis for the belief that primordial organisms had a very high degree of neutral genetic variability.

the intriguing hypothetical suggestion that the onset of parturition depends in part upon the maturation of trophoblastic antigens which then elicit from the mother the homograft rejection reaction.

Be this as it may, it is consistent with logic to postulate that the maternal immunologlical system as a selective agency is a decisive factor causing, in the mammals as compared to other vertebrates, a delayed epigenesis of a number of protein types and a consequent period of postnatal immaturity. Furthermore, the efficacy of this new selective agency in causing a delayed protein epigenesis is dependent on the degree of genetic variability in the species; the greater the genetic variability the more effective is the selective agency. For example, if an altered protein specificity has adaptive value to the species and if an allelic series of genes controls the differences in the time during ontogeny at which synthesis of the protein commences, the genes in the series which only begin to act in the postnatal organism will have a selective advantage over those which can provoke by their early ontogenetic activity a maternal immunological attack on the fetus. In a numerous species in the main stream of evolution, one which can inhabit a broad environmental zone, a sufficient number of alleles would be present in the population for such selection to occur. In the case of *Homo sapiens*, whose emergence occurred during the last million years, a time of extreme swings of climatic conditions, this process leading to delayed maturity might well have been speeded up if subspeciation during the reproductive isolation of the glacial periods alternated with hybridization during the migrations of the interglacial periods.

At this place in the discussion, it is worth drawing a distinction between the type of evolutionary change which leads to a phyletic advance and the type which is characterized by the processes of radiation and speciation. The former, progressive type of evolutionary change is one in which a more profound integration of the informational content of the new prototypic genotype leads to organisms with more extensive and complex ontogenies. In this type of change, the overall plasticity of the gene pool (the genetic variability) of the advancing population decreases, as pointed out above, while the differential between the greater degree of heterozygosity expressed by the ontogenetically late acting genes and the lesser degree expressed by the early acting genes increases. The latter radial type of evolutionary change is one in which a variety of somatic adaptations to narrow ecological niches are consummated after a sorting out of the initial genetic variability of the evolving population.

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In this type of change an emerging species in the dispersion remains at the same grade of phylogenetic development, and its population shows less genetic variability than the previous ancestral population. As differentiation continues and as the evolving species under intense selective pressure has to perfect its unique anatomical specializations to hold its position in its own narrow environmental habitat against mounting competition, its genetic variability will tend to be less than the variability of the contemporary phyletically advanced species which is still in the main stream of evolution. Even so, the genetic variability which may be greater in the advanced species will be centered in the ontogenetically late acting genes, not in the whole gene pool. Furthermore the total contemporary population extending through a number of genera at the lower phylogenetic grade of development will be expected to possess genetic diversities of greater depth than the total contemporary population at the higher phylogenetic grade of development, particularly on comparing the ontogenetically early acting genes of the two populations.

Le Gros Clark (1959) has succinctly summarized this distinction between radial and progressive evolution as manifested by the Primates by stating (p. 42) that "one of the outstanding features of Primate evolution has been, not so much progressive *adaptation* (as in other mammalian orders) but progressive *adaptability*." For example, to illustrate the point, the lemurs (an early offshoot from the primitive Primate stock) first flourished during the Paleocene and Eocene, at which time they dispersed over a wide geographical area. At a much later date in the Tertiary, when their accustomed habitats were being encroached upon by members of the Anthropoidea, certain of the lemurs succeeded in finding a sanctuary on the island of Madagascar and there radiated to give rise during the Pleistocene to a variety of constrasting genera and species, a number of which are still to be found on the island. On the other hand, the number of species among the most advanced of the Primates rapidly decreased during the Pleistocene. Indeed, as the hominids expanded from the jungles and savannahs to the farthest reaches of the earth, they became (and are yet becoming) more homogeneous in organic structure rather than less, and for the past 50,000 years have been represented solely by *Homo sapiens*, our own ubiquitous species.

The growing cohesiveness of the expanding population of hominids can be readily accounted for in terms of the thesis of this paper. Firstly, the maternal immunological system had operated as a selective agency

for a long enough time and with increasing effectiveness to ensure a sufficiently high basal level of genic homogenetity throughout the far flung hominid population to prevent the emergence of any serious deviations of the genotype during periods of reproductive isolation (especially since the beginnings of such deviation would also encounter the selective agency of the maternal immunological system). Secondly, the effects on ontogenetic development of this selective agency had helped create the circumstances by which the advancing hominids freed themselves of the confining territorialism of the other Primates. For, as pointed out at the beginning, a lengthening of the period of postnatal dependency, on the one hand, would provide just that formative psychological plasticity for the reshaping of "inborn habits" and the establishment of a more versatile intelligence and, on the other hand, would selectively favor the mutations which caused a progressive expansion in structure and function of the higher cortical centers of the brain. If we relate our thinking to that of Washburn and Avis (1958), it is not difficult to imagine how the hominids of the Pleistocene, with their increasing psychological plasticity, c ased to be habitual vegetarians and became opportunistic in their dietary habits. At first, more by chance than by design, they may have seized upon hard sharp objects and then used these objects to kill and dissect their game. But later they consciously turned to the use of tools and weapons as part of their mode of existence. Nor is it difficult to see that these hunters who roamed in bands over a broad geographical area must have learned to cope increasingly well with the large variety of environmental challenges that they met. With the disappearance of the need to adapt to a narrow habitat, the expanding population could maintain enough reproductive cohesiveness to circumvent the process of speciation. This, then, was a transitional stage of immense importance in the phylogenesis of living systems, after which the processess of cultural evolution, ushered in by man, could come to dominate further biological advance on the earth.

ENDOGENEOUS AND EXOGENOUS ENVIRONMENTS

A certain pattern can be discerned in the evolution of life from the first amorphous beginnings in nonliving substance to the present ascendency of highly organized individualized beings. Not only has the separation between the living world and the nonliving world become increasingly greater, but the differentiation between the inner environment and the outer environment has assumed a growing importance in

the phylogenesis of higher forms of life. With each phylogenetic advance, the self-contained integrity of the living system has become more thoroughly buffered from the disintegrating forces of the outside environment, and the intensity of the self-directed aspects of the existence of the system has become greater.

We have traced several major stages of this differentiation in the development of living matter. Our starting point, it may be recalled, was before the advent of oxygen in the atmosphere when our ancestors in Precambrian times were unicellular, relatively undifferentiated organisms who possessed, however, the same essential type of genetic apparatus found in organisms of the present day. This, then, was a stage in the evolution of life much later than the first beginnings, depicted by the coacervates of Oparin (1957) or the clay particles of Bernal (1951) or the microspheres of Fox (1960), when the first fluid systems had just begun to separate from the random agglomeration of inanimate substances found in the primeval seas. Although the evolutionary development of the genic mode of inheritance greatly increased the internal order of living systems, a great deal of passive heterogeneous composition was still manifested by our Precambrian ancestors due to the vast accumulation of neutral mutations in the gene pool.

In the next major stage that we dealt with, commencing with the advent of oxygen in the atmosphere, the living systems which evolved had the capacity to synthesize a greater variety of subtly structured proteins. Thus, among the phyletically advancing organisms, the number of neutral mutations which could occur decreased as phylogenesis progressed, since the probability increased that any one genic mutation would directly affect a physiologically active portion of the surface of a protein. Perhaps this stage of evolution in its pure form has its highest expression in the phylogenesis of the class Aves. Although the store of genetic variability held by any one modern Avian species must be quite formidable, it is likely, nevertheless, to be much smaller than that held by our ancestral species at the time the prototherian mammals were arising out of the therapsid reptilian stock. However, with the rise of the mammals, a new factor, the maternal immunological system, entered as a selective agency to limit gradually (and in time drastically) genetic variability within a population. A modern mammalian species, therefore, should not have as much overall genetic variability as a modern avian species. In the next major stage of evolution, the mammalian stage, there must also have been intermediate steps, of which each resulted

in a yet greater differentiation between the inner and outer environments of the progressively evolving organisms.

These intermediate steps may be characterized in terms of the degree of intimacy of the fetal-maternal union, and, related to this, the extent of protective care of the mammalian offspring during its developmental stages. It was, of course, the evolutionary trend to more effective nourishment of the developing embryo that inevitably caused the maternal immunological system to become a selective agency directed against the genetic variability of the evolving population. The very nature of this evolutionary trend sets in motion two antagonistic tendencies: one favoring survival by more effective nourishment of the offspring and the other harming survival by immunological aggression against the offspring. This evolutionary trend could continue in phyletically advancing populations only as the conflict between the two opposing tendencies was continually resolved by an increasing differentiation between the inner and outer worlds of the evolving organisms.

The significance of this proposition will be apparent if we consider the difference in the type of immaturity shown, for example, by the newborn opposum as compared with that shown by the newborn human. The living marsupials are representative of the ancestral mammals in which the above evolutionary trend first began. At this metatherian level of phylogenetic development there is little opportunity for an exchange of isoantigens between the embryonic and maternal circulations. Only a rudimentary chorio-vitelline placental connection is established during the very brief intra-uterine life of the embryo, and the young are born while still in an embryonic state. Embryogenesis is completed in the marsupial pouch, and the offspring, maturing rapidly, fend for themselves within a few months after birth. Compared with the human, this type of animal does not show nearly as much dissociation between the rates of maturation of the somatic systems which establish an endogenous environment and those which interact with the exogenous environment.

In contrast to the metatherian mammals, the eutherian or placental mammals have very elaborate gestational processes which perhaps reach a peak of efficiency in man. The early human embryo establishes an intimate placental connection deep within the uterine tissues, and throughout the long period of gestation maximal opportunity exists for an exchange of proteins between the fetal and maternal blood streams. It is noteworthy that the immaturity of the newborn human largely concerns those somatic systems, such as the immunological system, the higher

cerebral cortical centers, the neuromotor connections, and the reproductive system, that interact with the exogenous environment. On the other hand, the major components shaping the inner integrity of the organism, such as the viscera and the autonomic nervous system, do reach an advanced level of differentiation and maturity by birth. We have already concluded that the genes which act in prenatal development should show a high incidence of homozygosity throughout the human population whereas the genes which act primarily in postnatal development should show a relatively high incidence of heterozygosity. The prenatal organism can then obtain an optimum of nourishment from the exceptional intimacy of the fetal-maternal union and still not release the isoantigens which would provoke an immunological attack from the mother. Furthermore, there can even be the necessary increase in the genic polymorphisms which impart survival value to a population inhabiting a broad exogenous environment. For it is precisely the genes concerned with exogenous adaptations that will escape the selective action of the maternal immunological system by not expressing their information in protein synthesis until the postnatal phase of ontogeny.

It follows from the principles of our schema that the extent of the population variability of the prenatally acting genes sets a limit on how far the gestational processes can establish intimacy between the fetal and maternal tissues, for if this variability is high, a vast number of isoantigens will exist between each fetus-mother pair in the population, in which case too intimate a union will immunologically destroy the offspring. Therefore the intimacy of the fetal-maternal union must have increased in a stepwise manner, each new step being taken only after the preceding step had led to a proportionate decrease in genetic variability.

The relatively undifferentiated populations comprising the basal primitive stock ancestral to the present placental mammals probably contained within the average interbreeding unit a very high degree of genetic variability. However, during the process of radical evolution, each speciating population group would have lost some of the initial genetic variablility, due to selection against the allelic and mutant genes which were not specifically adaptive to the narrow environmental zone inhabited by the population and due to the reproductive spread of those genes which were. In this event the evolutionary acquisition of more advanced placental mechanisms more closely connecting fetal and maternal tissues may well have been obtained in one or another population group as a secondarily derived benefit of the process of speciation. Nor

would this evolutionary acquisition under these circumstances cause any further pronounced differentiation between the inner world and outer world of the organisms of the species concerned, even though it would cause a further reduction of the store of genetic variability in the species (permitting thereby a further evolution of placental mechanisms). In particular, we would not expect to see any marked prolongation of postnatal immaturity.

On the other hand, during the progressive evolution of the population continuum which now consists of human beings, the environmental zones inhabited by the phyletically advancing organisms selected for (rather than against) genic polymorphisms. In this situation, the increases in the intimacy between mother and child became the major factor leading to the decreases in the heterozygosity of the prenatally acting genes and, concomitantly, to a protracted epigenesis of certain protein types with the growing shift of genetic variability to the postnatally acting genes. The whole direction of phyletic change then began to center on the threat to survival imposed by the prolongation of infancy and the slowing in the offspring of the population of the maturational processes which prepare the individual for the challenges of the outside world. The danger to the population was overcome, of course, by the evolutionary development of family and social life, again on the basis of an intimacy between mother and child, only this time an emotional intimacy rather than an organic one. The outgrowth from the mammalian stage of evolution of the qualitatively new stage of cultural evolution originates, therefore, in a complex of events involving at the start immunochemical differences between individuals.

From the perspective of our theory, we can readily accept the conclusion which Le Gros Clark (1959, p. 314) draws from the comparative studies of contemporary Primates that "the progressive differentiation and elaboration of placental mechanisms, which evidently promote a more and more efficient means of securing the optimum conditions for the developing embryo, comprise one of the most outstanding features of Primate evolution." The premise for this conclusion derives from the opinion of embryologists (see, *e.g.*, Hamilton *et al.*, 1952), based to some extent on nutritional considerations, that the epitheliochorial placenta represents the primitive placental arrangement of the basal ancestral stock of eutherian mammals, whereas the hemochorial placenta represents a far more advanced arrangement that arose at a later stage of evolution. We can support this opinion concerning the evolutionary

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sequence of placental mechanisms with reasons that are compelling on immunological grounds. The epitheliochorial type of placental arrangement (consisting of two avascular cellular layers in addition to the linings of the fetal and maternal blood vessels) forms a firm barrier to the exchange of isoantigens between the fetal and maternal circulations and therefore appears to represent the primitive arrangement. On the other hand, the hemochorial placenta (in which fetal and maternal circulations almost touch) forms a very weak barrier to the exchange of such isoantigens. Thus the hemochorial placenta could only become fully developed without catastrophic immunological consequences after the high genetic variability of the ancestral population had been sufficiently reduced. The contemporary Primates arranged according to their level of phylogenetic development show placental mechanisms (described, in particular, by Hill, 1932) which progressively increase the actual intimacy of the union betwen fetal and maternal tissues.

The lemurs have an epitheliochorial placenta and thus appear to have retained a genuinely primitive type. This could help explain in terms of our theory the fact, already discussed, that at a late stage in lemuroid evolution an explosive radiation occurred out of which emerged a number of new contrasting species. Due to the primitive placenta, the maternal immunological system of the lemurs could have acted only in a weak manner to limit the genetic variability of the evolving populations. There is, however, a tendency in the lemurs to the placental arrangement distinctive of the higher Primates, in that the chorion differentiates early and becomes precociously vascularized. This tendency is even stronger in the genus Galago. Furthermore, the tree shrews and Tarsius each have a hemochorial placenta, the one in Tarsius closely approaching the type found in the Anthropoidea. The significant feature of placentation which concerns us in this discussion is that more and more tissue systems of the developing embryo become accessible to the selective action of the maternal immunological system in the following series: prosimians, platyrrhine monkeys, catarrhine monkeys, and finally anthropoid apes and man. The establishment of a functional placenta by the developing embryo becomes ever more rapid, and the vascularization of the chorion becomes ever more precocious. For example, as previously mentioned, the embryo of man while still exceedingly small burrows deep within the uterine wall and gains a most intimate relation to the maternal blood stream. In all the lower Primates, on the other hand, much of embryonic development occurs in the lumen of the uterus

before an intimate union with maternal tissue is established. Thus there would be less opportunity in man than in the lower Primates for isoantigens to form in the developing embryo at tissue sites sufficiently hidden from the maternal blood steram that the danger of isoimmunizing the mother is removed. This danger, of course, has been minimized during the progressive evolution of the Primates by a weeding out of isoantigens and by a delayed epigenesis of a growing proportion of the remaining ones.

IMMUNOLOGICAL TOLERANCE AND AUTOSENSITIVITY

The assumption that the intimate union of fetus and mother in the higher Primates has had far reaching consequences of an immunological nature receives indirect support from the few comparative studies so far carried out (reviewed by Billingham, 1958) concerning immunological tolerance. Of the mammals studied, the members of those groups (*e.g.*, rabbit, dog, and rat) with relatively intimate fetal-maternal placental arrangements had tolerance responsive periods to homografts which extended through the prenatal stage of development not terminating until one to 14 days after birth, depending on the species. Even in man, whose intra-uterine existence is of a much longer duration, the tolerance responsive period may last until the time of birth. On the other hand, fetal sheep lost this ability to acquire immunological tolerance to homografts at least 50 days before birth (Schinkel and Ferguson, 1953), and fetal cows also lost this ability long before birth (Billingham and Lampkin, 1957). The homografts in these fetal animals provoked an immune reaction and were rejected. The Artiodactyla, of course, have the epitheliochorial placenta which (as already suggested) erects a firm barrier to the exchange of antigens between mother and fetus.

Thus, according to our view, a fetal ungulate can reach, while still in utero, an advanced stage of antigenic maturation without provoking a maternal immunological attack. Similarly, after this stage is reached, the mesenchymal progenitors of the immunological system, having become sufficiently acclimatized (tolerant) to a host of potential autoantigens, can begin to mature long before birth without harmful immunological consequences (though the complete maturation of the immunological system with its various types of intracellular and circulating components may take a considerable period of time).

In the other mammalian groups in which there is intimate fetal-maternal union, the fetus will be subjected to a crippling maternal

immunological attack if its prenatal maturation becomes too extensive (unless, of course, a very high degree of intraspecific antigenic homogeneity characterizes the population group). Similarly, if the genes controlling antibody synthesis were activated prenatally the fetus could immunologically attack its maternal host and also respond in a self-destructive manner to its own developing antigens. For these reasons, the maturation of the immunological system must be delayed throughout prenatal life, and in fact is so delayed as shown by the studies cited above.

A critical situation must now inevitably develop after birth. The neonatal organism cannot resist for long microbial invasion unless its own immunological system rapidly matures. Yet, once the antibody synthetic mechanisms are activated, the potential for self-destructive autoimmune reactions also arises, since immunological tolerance will be lacking for those endogenously produced protein substances as well as those exogenously introduced substances which have never had a prior systemic contact with the fetal mesenchymal progenitors of the immunological system. Human beings with their pronounced delayed epigenesis of isoantigens are especially confronted with the danger of autodestructive immunizations. We may predict that there have been at least several evolutionary developments by which this self-destructive tendency has been held within tolerable bounds. These devlopments which are considered below concern: (1) an inaccessibility to the blood circulation of isoantigens and autoantigens, (2) physiological mechanisms for repressing immunological reactivity, and (3) the process of fetalization.

The first development relates to the increasing differentiation, with progressive evolution, of the endogenous environment of the living system. Those mutant genes and their heterozygous combinations which give the living system a greater capacity to resist the disintegrating impact of the exogenous environment will have an added selective advantage if their information can be expressed solely in molecular structures that are insulated from the antibody producing cells. Such structures can be isoantigenic and autoantigenic, can have markedly delayed epigenetic histories, and still can escape the destructive immunological consequences of their late appearance. In this essentially blind manner, the immunological system as a selective agency acts through autoimmunizations as well as through maternal isoimmunizations to increase the complexity of macromolecular organization within

the surviving individuals of the phyletically advancing species. For example, the progressive evolutionary expansion of the brain, an organ known to contain isoantigens and autoantigens, probably occurred under these conditions of relative inaccessibility to the immunological system. Indeed, considering the opportunistic nature of evolutionary advance, it follows from our thesis that the central nervous system, due to its pre-existing insulation from the antibody producing cells, could take greater advantage than many of the other tissue systems of the selection in the mammals for ontogenetically late acting genes.

The second evolutionary development concerns particularly those potentially antigenic substances synthesized under the control of ontogenetically late acting genes, which do have various degrees of accessibility to the immunological system. Though many of these substances may not normally circulate, we can expect that some of them will be released from their tissue sources after an injury or during severe muscular activity. Under these conditions of stress, the organism would be greatly benefited by having physiological mechanisms which act to depress inflammatory reactions and lessen the sensitivity of the immunological system. As a matter of fact, precisely such mechanisms do come into play in the well known hormonal response to stress (Selye and Heuser, 1956), for the increased secretion of ACTH following stress causes more intense adrenal cortical activity and thereby an increase in the blood level of the anti-inflammatory steroids such as hydrocortisone. At pharmocological levels these steroids drastically repress the functioning of the immunological system. At the physiological levels involved in the ordinary response to stress, the diminution of antibody synthetic capacities is probably not large. Nonetheless, all that may be necessary is a transitory, moderate lessening of the sensitivity of the immunological system while the released tissue antigens are being metabolized.

These tissue antigens probably have only weak antigenic properties within the organism. Assuming that their macromolecular structures are largely shaped by the time of birth, their autoantigenicity would reside only in minor structural changes consummated during the postnatal maturational processes of the organism. The contention that, after a small lessening of the immunological responsiveness of the organism, these substances normally would not provoke autoimmune reactions receives indirect support from the studies on tolerance of the Y factor in C57 mice (Billingham and Silvers, 1960). This factor, under the control of a locus on the Y chromosome, is the histocompatibility

antigen responsible for the rejection of male skin isografts by female mice which, of course, lack the Y chromosome. The crux of our argument lies in the following observations. Firstly, on comparison with other histocompatibility antigens, the Y factor is only weakly isoantigenic. Secondly, the immunological system of the new born mouse matures fairly rapdily after birth, the tolerance responsive period to homografts ending by the second day. Yet for this Y factor, which simulates, as Billingham and Silvers (1960) suggest, a weak autoantigen, the tolerance responsive period lasted until 17 days after birth. Thus, to restate our contention, if the immunological system is not completely responsive (whether due to incomplete maturation as in the case of the tolerance experiments with the Y factor, or whether due to stress as in our hypothetical example) a weak antigen will not provoke an immune response whereas a strong antigen will.

The claim that the physiological changes elicited by stress involve a weakening of immunological responsiveness is supported by the report of Wistar and Hildemann (1960) that chronic avoidance learning stress in mice depressed the immune reaction responsible for skin homograft rejection to a modest but significant degree. While implicating the adrenal corticoids, these authors do not rule out the possibility that other as yet unknown channels may mediate the stress-induced inhibition of the homograft rejection reaction.

We may also suppose that adaptive mechanisms for repressing antibody production come into play to meet the challenge of those freely circulating macromolecules which undergo epigenetic changes long after the immunological system is functional. Again the potential autoantigenicity of these substances would ordinarily be weak and would reside in new isoantigenic configurations introduced by the maturational changes in the activity of genes from the paternal haploid contribution. For example, Goodman (1960a, b) has observed that there are subfractions of human gamma$_2$ globulin which vary in specificity between human beings and which are synthesized more frequently after puberty than before puberty. We would expect that if an individual produced the same isoantigenic subfraction as his mother, he would have acquired an initial immunological tolerance to this fraction as a result of the previous prenatal transfer of maternal gamma globulin into his blood stream. On the other hand, if the isoantigenic subfraction is not one possesed by the mother, it should be a potential autoantigen. Apparently, the body adapts to this type of stress and ordinarily becomes immunologically

unresponsive to such isoantigens. In fact, immunologically functional animals injected with large quantities of frankly foreign protein do acquire in some instances a state of immunological unresponsiveness to the injected material (Dixon and Maurer, 1955). However, it is clear that too intense a repression of immunological responsiveness will impair survival chances by causing an increased susceptibility to infections. Therefore the potential autoantigens which can be tolerated in blood must be few in number (as compared to those immobilized in tissues) since such circulating entities impose a mounting burden on the body even in the absence of autoimmune reactions.

An inference that can be drawn from the above reasoning is that during the evolutionary rise of the higher mammals (particularly, man) the triggering of the physiological mechanisms for the repression of immunological responsiveness has come increasingly under the influence of higher centers of the brain, their pertinent signals acting through the hypothalamus. It would be adaptive for the higher centers to release such signals as part of the preparation for meeting the attack of hostile macroorganisms or for pursuing game, in other words, under the primitive conditions of stress when autoantigens are likely to enter the blood stream. Such signals would not be released by the invasion of microorganisms and, indeed, during periods of rest, these signals would be inhibited so that full immunological responsiveness to microorganisms could be attained.

Fetalization or neoteny, a characteristic process in the evolutionary development of man (Bolk, 1926; de Beer, 1940), provides yet a third means of circumventing the destructive effects of iso- and autoantigens. According to de Beer (1940), one of the more striking cases of neoteny is the progressive loss of body hair in the higher Primates. For example, the body of the monkey at birth is completely covered with hair. Yet only the head and back of the newborn gibbon have hair; the other body regions of the gibbon are covered later. The gorilla at birth has hair only on his head, and his other body regions become partially covered later on. Man, too, is born with hair on his head, but in contrast to the gorilla very little hair appears on the rest of his body throughout his life. Let us suppose then, to illustrate our thinking, that in the primitive Primates the maturation of the integumentary cells that manufactured hair also gave rise (perhaps due to linked polygenic arrangements) to a number of isoantigens. If so, maternal immunization to these isoantigens as a result of an increasing fetal-maternal

intimacy in the progressively evolving population group caused (according to our supposition) a selection for genes that delayed the full maturation of the hair cells until after birth. Then, as evolutionary advances continued, autoimmunizations due to the delayed maturation of the antigens associated with these genes contributed to the selective pressures which brought about in the most advanced Primates a lasting state of immaturity in respect to body hair. Finally, the use of tools and the creation of language and culture allowed our predecessors who survived the last glacial age to live in the arctic northlands despite their hereditary nakedness. If we continue through science and its social application to gain an ever growing dominance over the environment of the earth, there is reason to believe that fetalization as a means of resolving our self-destructive immunological antagonisms will become yet more decisive in our future evolutionary development.

PERSPECTIVES FOR RESEARCH

By considering the role of maternal immunizations and autoimmunizations in the phyletic development of human behavior, we have, in effect, been attempting to integrate certain of the factual and conceptual matter of immunology into the broader framework of evolutionary theory. This attempt at integration will have merit if it discloses connections between otherwise independent observations and if in leading to predictions which can be subjected to the test of experiment it opens up new areas for research.

Our general thesis and the relationships derived from it are in fact supported by observations concerning the maturation of isoantigens and autoantigens. For example, the histocompatibility antigens appear to be less well formed and show less individuality in embryonic skin than in adult skin. Homografts of human embryonic skin on adults are claimed to be successful[4] (Toolan, 1958; Snyderman, 1958), but homografts of adult skin have always been failures. This suggests that the epigenesis of the histocompatibility antigens has been strongly influenced by the maternal immunological system as a selective agency.

The results of blood group serology also relate to our general thesis. Assuming that the blood group polymorphisms reflect beneficial com-

[4] Other opinion holds that such fetal grafts will not survive indefinitely. Nevertheless they do survive much longer than adult grafts (*e. g.*, Baxter, *et al.*, 1958) demonstrating thereby the incomplete nature of histocompatibility antigens in fetal skin.

binations of heterozygous genes, there will be added advantage to those genes which fail to express their information strongly in prenatal development and thereby escape the selective action of the maternal immunological system. It is of some interest, therefore, that the A and H antigens of human red blood cells appear to undergo a further maturation after birth and, after a number of months, become much more readily detectable by the ordinary serological reagents (Dr. Charlotte Otten, unpublished data). We would expect that this tendency of blood group antigens to undergo a protracted epigenesis has been accentuated in the Primates at each higher stage of phylogenetic development. We could learn from comparative studies whether or not this tendency was accentuated in the predicted manner.

The experimental auto-allergic diseases also provide cases, this time concerning autoantigens, which support our general thesis. For example, the causative antigenic fraction of experimental allergic encephalomyelitis is not present in fetal brain but is present in adult brain (Kabat *et al.*, 1947). Similarly, the causative testicular antigenic fraction of experimental allergic aspermatogenesis matures quite late in ontogeny (Isojima and Stepus, 1959; Katsh, 1960). The causative organ-specific factor appears to be much more species specific than the factor in experimental allergic encephalomyelitis (Freund *et al.*, 1953; Paterson, 1959). As more autoantigens are discovered, particularly those involved in spontaneous clinical disease such as certain of the thyroid proteins (*e.g.*, Roitt *et al.*, 1958; Holbrow *et al.*, 1959), it becomes important to determine their phylogenetic and ontogenetic histories. We would want to know, for example, the basis for the difference in species specificity between the thyroid antigens involved in spontaneous clinical diseases (such as Hashimoto's disease) and those additional antigens involved in the induced experimental disease. The autoantibodies directed to the former show a high degree of species specificity (Roitt *et al.*, 1958) cross reacting only with thyroid extracts of the higher Primates, not with thyroid extracts of other mammals. On the other hand the induced antibodies to thyroid extracts cross react broadly with mammalian thyroids (Witebsky and Rose, 1956; Rose *et al.*, 1960). We would want to know if this difference in species specificity can be related to a difference in the epigenetic histories of the antigens concerned. Our theory predicts that there is a tendency for the naturally occuring autoantigenic substances to show a delayed maturation of their antigenic potency in the mammals, especially so in the higher Primates, and that they should thereby have more autoantigenic potency in the more phyletically advanced organisms.

The predictions of our theory can also be tested by comparative studies of the immunologic species specificities of the serum proteins of the Primates. We are engaged in such studies (Goodman, 1960c) and these continue to disclose relationships in accord with the theory. For example, with a variety of antisera to human serum albumin we have consistently failed to demonstrate any specificity differences between the albumins of man, gibbon, and chimpanzee (the only hominoids tested so far). Yet with our antisera to human gamma globulin we were able to distinguish the human protein from its counterparts in gibbon and chimpanzee, the evolutionary divergencies of gamma globulin being greater between man and chimpanzee. Albumin, of course, arises early in embryogenesis and is a key constituent of the endogenous environment. Gamma globulin, on the other hand, shows a marked epigenesis in postnatal development and is a key constituent in meeting the hostile challenges of the exogenous environment. The failure of albumin to show evolutionary divergencies in the hominoids can be readily explained in terms of the increasing effectiveness of the maternal immunological system as a selective agency directed against genetic variability. Since gibbon-like hominoids appear to have begun differentiating from the main hominoid stock in the order of 40,000,000 years ago (Le Gros Clark, 1959), we can see that our postulated selective agency (or its equivalent) has been a truly powerful force in the phylogenesis of the higher Primates.

Even in the lower Primates our preliminary data indicate that this agency has been at work. For example, as previously mentioned, the galagos, more so than the lemurs, show some approach to the higher Primates in the intimacy of fetal and maternal tissues during gestation. In correlation with the role attributed to maternal immunizations, we observed that on comparing the sera of Lemur and Galago with antisera to human albumin the Galago albumin yielded a stronger cross reaction than the Lemur albumin. We are anxious to gather a more inclusive collection of prosimian sera and study their relationships in more detail with antisera produced directly against their proteins. According to our theory, the lemurs (especially due to their epitheliochorial placenta) should show antigenic divergencies of their albumins at a much lower taxonomic level than the hominoids. Similarly, monkeys should also show such divergencies, though to a lesser extent. In this connection, although none of the antisera to human albumin detected any differences in the albumins of different families of hominoids, some of these same antisera have detected differences in the albumins of the cercopithecoids at the subfamily level and also of the ceboids at the subfamily level.

For example, Wilson[5] observed with an anti-human albumin sera that langur could be distinguished from baboon and capuchin monkey from spider monkey (Fig. 1).

Certain relationships predicted by our theory also appear to be emerging from another set of data on Primate sera that we are gathering by a combination of starch gel electrophoretic and immunological techniques. Extending the results of Blumberg (1960), we are finding (Fig. 2) in the genus Macaca a striking polymorphism of transferrin among rhesus monkeys (*Macaca mulatta*) from India, 15 patterns of this iron-binding protein having been detected in the 77 monkeys examined (13 patterns alone in 40 monkeys captured in one small locale near the border of Nepal). On the other hand, we have detected only a single transferrin type among cynomolgus monkeys (*Macaca irus*) from the Philippines (79 of these monkeys have been examined). Let us assume on the basis of these observations that the environment of *Macaca mulatta* promotes genic polymorphisms, whereas the environment of *Macaca irus* (Philippine variety) selects against genic polymorphisms. Obviously, this is an assumption whose generality so far as the non-transferrin loci are concerned has to be tested by further experiments. Its plausability lies in the fact that the environment of India contains many more ecological zones than the environment of the Philippines. It then follows that the rhesus monkey, as a polymorphic of heterozygous animal, should synthesize many more potential autoantigens than the cynomolgus monkey, in which case the physiological mechanisms which have evolved for repressing the sensitivity of the immunological system should be much more active in rhesus monkeys than in cynomolgus monkeys. As a matter of fact, in support of our reasoning, we have observed that the mean serum gamma globulin level is only 11.1% in rhesus monkeys as compared to 17.7% in cynomolgus monkeys. This species difference in gamma globulin was found to be significant at the 0.1% level of confidence by the t test.

We have also discovered (Goodman and Poulik, 1960; Goodman *et al.*, 1960) that the analysis of primate sera by two-dimensional zone electrophoresis (filter-paper electrophoresis in length followed by starch-gel electrophoresis in width) yields protein patterns which accurately reflect accepted evolutionary relationships among the Primates, especially at the lower taxonomic levels. Examples of these protein patterns are presented in Fig. 3. It can be seen that the patterns of the Cercopithecoidea are sharply distinct from those of the Hominoidea and that there is less

[5] Dr. Morris Wilson, deceased, 1959.

variation in pattern between members of the subfamily Cercopithecinae than between members of the superfamily Hominoidea. The interspecific variation in the genus Macaca is minimal. Our theory predicts that the interspecific variation of pattern at the genus and family levels should be much greater among the lower Primates than among the higher Primates. We are anxious, therefore, to study by two-dimensional zone electrophoresis the protein patterns of the Prosimii as well as the Anthropoidea. This study and the one involving a comparative analysis of the antigenic divergencies of primate proteins should eventually enable us to view the evolutionary position of man in clearer perspective.

SUMMARY OF THE THEORY

Progressive evolution and radial evolution have been manifested by the Primates during their phylogenesis. Progressive evolution carried part of the primate population to higher stages of phyletic development. At each stage radial evolution split up the remainder of the population into speciating units that failed to advance phyletically. The environment selected for heterozygotic advantage (polymorphisms) in the progressively evolving group and for homozygotic adaptation in each speciating unit. One population advanced into a broader environmental zone. The other population, radiating out of the first, fragmented into separate speciating units, each perfecting adaptations to a contracting environmental niche. The gene pool of the first population approached the condition described by Dobzhansky (1955, 1960) in his "balanced hypothesis" in which the individuals heterozygous for genes had greater fitness. The gene pool of each diverging unit of the second population approached the condition ascribed by Dobzhansky to the "classical hypothesis" in which the individuals homozygous for genes had the greater fitness. At the same time, the genetic diversities which ultimately arose between the diverging units of the radiating population were of much greater depth than the genetic diversities which existed among the members of the phyletically advancing population.

On the basis of considerations concerning protein structure, we were able to postulate that the basal ancestral stock of organisms out of which these populations evolved had a large measure of genetic variability, but one which neither favored survival nor worked against survival, and was thereby a reservoir for preadaptive evolutionary changes. It was essentially under these genetic conditions that the sustained evolutionary trend in the mammals to more effective nourishment of the developing

FIG. 1. CROSS REACTIONS OF PRIMATE SERA WITH A RABBIT ANTISERUM (R-226) TO HUMAN SERUM ALBUMIN (HSA). Highly sensitive Wilson Comparative Analysis Plates of triangular design were employed. The mold in each plate was filled with molten agar, and after the agar congealed the basins at the base and sides of the triangle were cut out. The antiserum was placed in the bottom basin and a different Primate serum (antigen) in each of the two side basins. Antibody and antigen molecules diffused into the agar and formed a precipitate (the dark line) when they reached each other.

In (a) the gibbon line and the human line merged, yielding the reaction of identity. This means that no difference in human albumin and gibbon albumin was detected.

In (b) the rhesus albumin yielded a reaction of only partial identity with the gibbon albumin, the gibbon line extending beyond the meeting point with the rhesus line to form a spur. This means that antibodies to certain of the configurations of human albumin failed to react with any of the configurations of rhesus albumin but did react with configurations of gibbon albumin.

In (c) the rhesus albumin yielded a larger cross reaction than the spider monkey albumin, the rhesus line yielding the spur.

In (d) the langur albumin and the gelada baboon albumin yielded reactions of partial identity with each other, each line yielding a spur.

In (e) the mandrill albumin yielded a larger cross reaction than the marmoset albumin, the mandrill line yielding the spur.

In (f) the spider monkey albumin yielded a larger cross reaction than the capuchin albumin, the spider monkey line yielding the spur.

These photographs of results obtained by the late Dr. Morris Wilson are reproduced with the kind permission of Mrs. Mira Wilson.

FIG. 2. A. COMPARATIVE ONE-DIMENSIONAL STARCH-GEL ELECTROPHORESIS OF FIVE RHESUS SERA (LEFT) *and* FIVE CYNOMOLGUS SERA (RIGHT). The Roman numerals printed below the photograph of the gel designate the transferrin types of these samples. Note the uniformity of the cynomolgus transferrins (type I) and the variability of the rhesus transferrins (types II, XII, XI, VI, IX).

B. COMPARATIVE ONE-DIMENSIONAL STARCH-GEL ELECTROPHORESIS OF 15 RHESUS SERA REPRESENTING THE 15 TRANSFERRIN TYPES. Below the photograph of the gel, that portion of the photograph showing the transferrin bands is presented diagramatically, with the number of each type found among 77 rhesus monkeys.

Trans. = transferrin.

FIG. 3. PROTEIN PATTERNS OBTAINED BY TWO-DIMENSIONAL (FILTER-PAPER IN LENGTH, STARCH-GEL IN WIDTH) ELECTROPHORESIS OF HIGHER PRIMATE SERA. Representing the superfamily Hominoidea are man (E-633), chimpanzee (E-680), and gibbon (E-676). Representing the subfamily Cercopithecinae are cynomolgus monkey, *Macaca irus* (E-96), rhesus monkey, *M. mulatta* (E-160), pigtailed macaque, *M. nemestrina* (E-726), and baboon, *Papio papio* (E-86).

For orientation, gamma globulin (γ glob.), transferrin (trans.), and albumin (alb.) are labeled, and for emphasis the components of the patterns are outlined on the photographs of the starch-gels.

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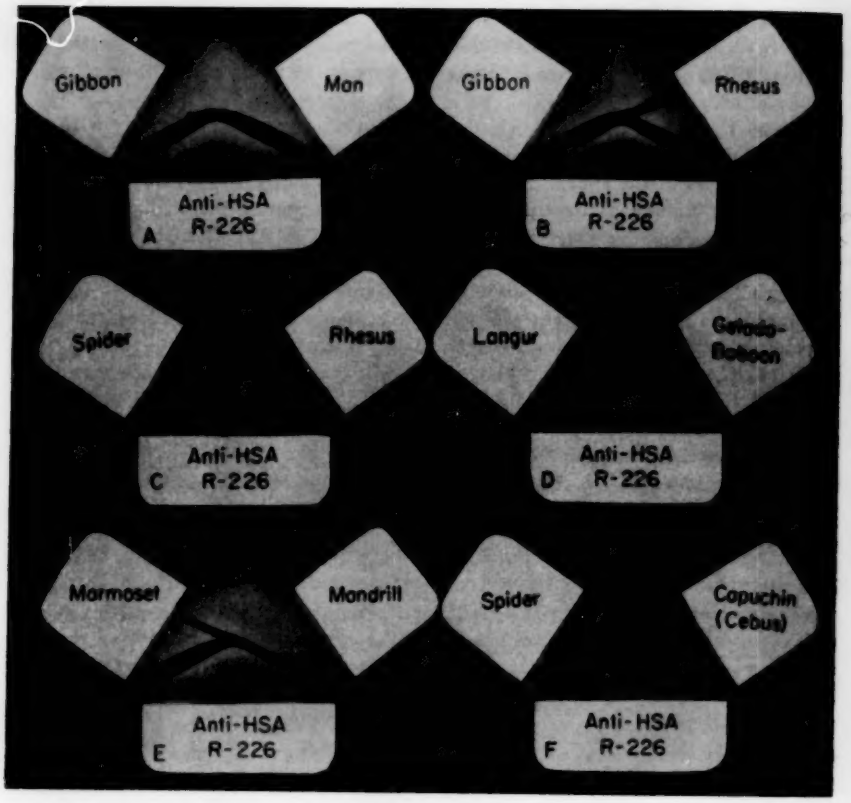
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Anti-HSA
R-226
C
Anti-HSA
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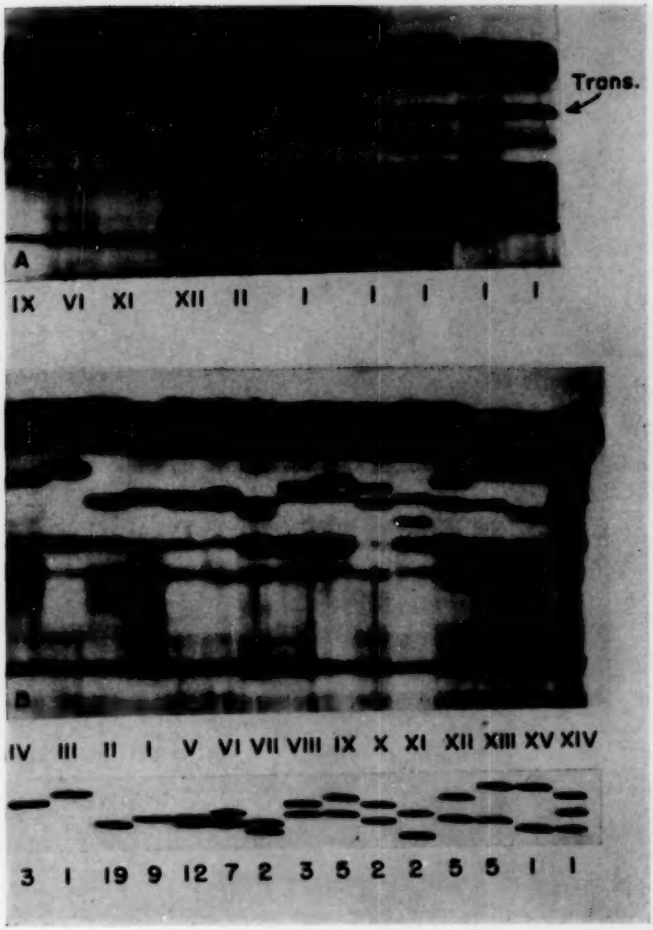

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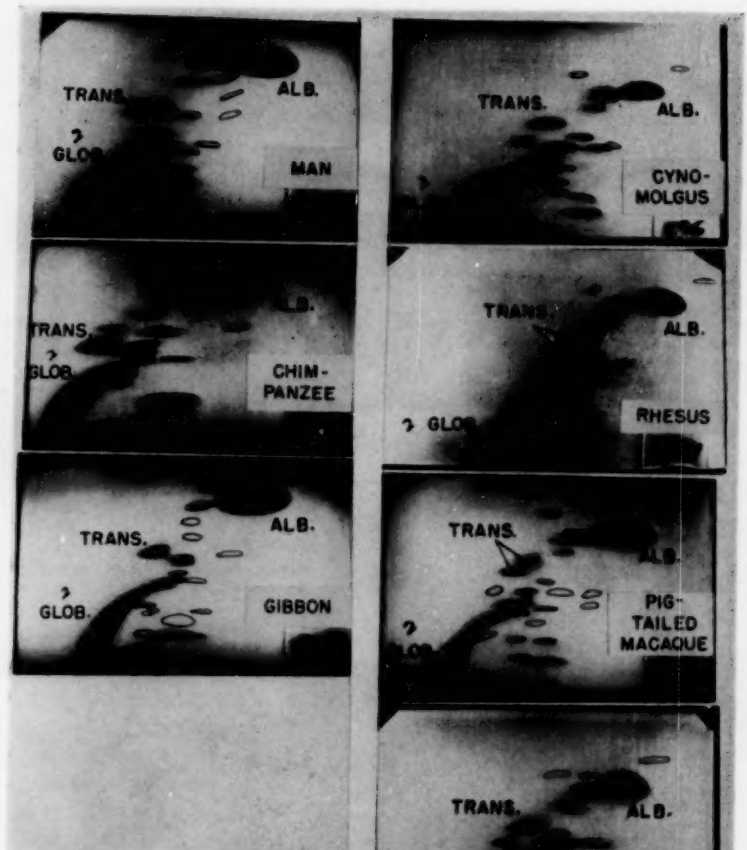
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embryo introduced the maternal immunological system as a new and powerful selective agency directed against genetic variability. Crisis after crisis inevitably arose in the progressively evolving population in which selection for heterozygous combinations of genes by the environmental zone conflicted with selection against these combinations by the maternal immunological system. Phyletic advance resulted when each such crisis was resolved in a way that led to a dissociation within the evolving organisms of the rates of maturation of those systems which primarily establish the endogenous environment and those systems which meet and overcome the hostile challenge of the exogenous environment. The first systems still matured prenatally but became more uniform throughout the population, avoiding by this means attacks from the maternal immunological system. The second systems, on the other hand, were immature at birth. Their antigenic constituents showed a protracted epigenesis and became during postnatal development the center of genic polymorphisms but also potential autoantigens (in view of the rapid neonatal activation of the immunological system). The repression of immunological responsiveness by physiological mechanisms that can be triggered by brain signals when an organism is under stress then became more important as evolution continued.

We have argued that the tendency to protracted intimacy between mother and child could find a powerful expression in the Primates, for under the selective pressures of an arboreal environment the Primates had early in their phylogenesis begun to show a progressive expansion of the brain. In turn, the very expression of this tendency, while disrupting "inborn habits," created additional selective pressures for a further development of those somatic systems which underlie the psychological plasticity and versatile intelligence of the higher Primates. Simultaneously, as a result of the ever increasing effectiveness of the maternal immunological system as a selective agency, the overall plasticity (variability) of the gene pool decreased. The dynamics of the process led to a phyletically advancing population which ceased to speciate. For the first time, behavior patterns had been shaped which liberated the members of a population from the complete domination of the environment. This signified the advent of the current cultural stage in the phylogenesis of life.

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ON THE RELATIONSHIP OF SCORES ON CERTAIN PSYCHOLOGICAL TESTS WITH A NUMBER OF ANTHROPOMETRIC CHARACTERS AND BIRTH ORDER IN TWINS

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INTRODUCTION

THE idea that the psychological attributes of normal human beings are to some extent a function of their physical dimensions is very old. The idea is *a priori* reasonable and is widely applied in human relations, but it has become largely discredited by the failure of most attempts to demonstrate the existence of such relationships in quantitative terms. A few investigators, notably Pearson (1902, 1906, 1925) and Pearl (1906) have reported very slight positive correlations between intelligence and head size. These corrleations were so low, however, that within the normal range of variation the anthropometric and psychometric traits are usually assumed to be essentially independent of one another.

It is possible, however, that the failure to demonstrate any appreciable relationships between these two sets of variables is due not to the absence of relationships between them but to the cultural and genetic heterogeneity of the individuals studied. Psychometric characters are affected by so many factors, both environmental and hereditary, that relationships between them and the anthropometric traits might be difficult to demonstrate even if they existed in considerable magnitude. If this argument is correct, one should not expect to find significant cor-

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relations between anthropometric and psychometric traits in random samples from the general population unless these samples are very large.

In the present investigation of relationships between anthropometric and psychometric variables the effects of extraneous environmental and genetic differences have been minimized by basing comparisons on differences within pairs of like-sexed twins. The use of twins in this way provides a particularly sensitive method of investigating interrelationships among highly variable traits and might be expected to reveal relationships which are too small to show up in samples from the general population.

ACKNOWLEDGMENTS

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MEASUREMENTS

The scores on three psychological tests are included in this study, namely the *Progressive Matrices Test*, the *Chicago Test of Reasoning Ability*, and the *Chicago Test of Verbal Ability*. The Progressive Matrices Test was constructed by J. C. Raven (1938, 1941) as a nonverbal test intended to measure Spearman's general intelligence factor, "*g*." The Chicago Test of Reasoning Ability and the Chicago Test of Verbal Ability are two of six parts of the Chicago Tests of Primary Mental Abilities, which were devised by L. L. Thurstone and T. G. Thurstone as measures of the six most important distinguishable aspects of intelligence (L. L. Thurstone, 1938; L. L. Thurstone and T. G. Thurstone, 1941a, 1941b). The three mental tests chosen for this study are

believed to be measures of the most important kinds of cognitive skills. Since it appeared impractical to consider all of the many mental tests administered by the Hereditary Abilities Study it was decided to concentrate first of all on those cognitive skills which are generally regarded as most important. In addition to the three psychological tests mentioned above a function of them called *Total Test Score* and defined as the sum of the scores on the three separate tests also was investigated in relation to the anthropometric characters and birth order. When a psychological test was lacking for one or both of the co-twins in a pair the Total Test Score was obtained from only those tests which were given to both twins.

The anthropometric measurements included in the study are *Head Length* (1), *Head Breadth* (3), *Bizygomatic Breadth* (6), *Head Circumference* (45), *Stature* (1), and *Weight* (71). The definitions of these characters and the methods employed in making the measurements are those of Martin (1928), who designated the various traits by the "Martin Numbers" shown in parentheses. Weight was measured to the nearest pound; the other physical measurements were taken to the nearest millimeter. In addition to the direct measurements enumerated above, three functions of certain of these measurements also were studied, namely *Cephalic Index*, defined as Head Breadth divided by Head Length, *Reciprocal Ponderal Index*, defined as Stature divided by the cube root of Weight, and *Index of Cross-Sectional Head Area*, which is defined as Cephalic Index times Head Circumference. The Index of Cross-Sectional Head Area was suggested by Philip Eckstrom; it is a more reliable index of general head size than is Head Circumference taken alone, since the actual cross-sectional area of the head is a function of both Head Circumference and Cephalic Index. It would be desirable to have an index of head volume also, but unforunately our measure of head height is not sufficiently accurate to be used for this purpose.

Birth Order is also regarded as a measurement, the first and second born co-twins being designated *a* and *b*, respectively. This measurement is not applicable to one pair of monozygous females that was delivered by caesarean section.

For convenience in tabling and analyzing the data the 14 characters are coded as follows:

M: Progressive Matrices Test
R: Chicago Test of Reasoning Ability
V: Chicago Test of Verbal Ability
T: Total Test Score
L: Head Length
B: Head Breadth
Z: Bizygomatic Breadth
C: Head Circumference
S: Stature
W: Weight
I: Cephalic Index
E: Index of Cross-Sectional Head Area
P: Reciprocal Ponderal Index
O: Birth Order

TWINS

Measurements were taken on a series of 20 female and 23 male monozygous twins and 23 female and 14 male like-sexed dizygous pairs. The measurements for these twins are given in table 1, with the values for the first born co-twin in each pair being given first. Certain measurements are missing for some individuals. Except for three pairs who were undergraduates at the University of Michigan, the twins were, at the time the measurements were made, from the junior and senior high schools of Ann Arbor, Ypsilanti, Dearborn, and Detroit. The twins ranged in age from 12 to 20 years, the median age being 16.

Twins were classified as dizygous if (1) they were discordant in either the ABO, MN, Rh, Kell, Duffy, or secretor reactions, if (2) the color or pattern of the iris of their eyes were conspicuously different, or if (3) their facial and head characters—including hair color—were markedly dissimilar. Otherwise they were classified as monozygous. The ABO types were based on the antisera A, absorbed A, and B; the Rh types were based on the antisera C, D, E, c, and e.

METHOD OF ANALYSIS

The relationship between any two traits, X_i and X_j, was investigated by comparing the signed difference between co-twins, $a - b$, in trait X_i with the corresponding difference in trait X_j for each pair of twins.

A method of detecting relationships between variables which is relatively free from restrictive assumptions involves only the sign of the

TABLE 1

Basic data upon which this study is based. The first born co-twin is listed first for each twin pair. See text for code of measurements

Monozygous females 1A

Number	M	R	V	T	L	B	C	I	E	Z	W	S	P
16572	48	53	66	167	183	140	522	.77	399	120	103	1682	359
16573	35	42	61	138	188	138	535	.73	393	123	108	1699	357
16579	47	69	88	204	189	137	542	.72	393	129	118	1620	330
16580	53	74	84	211	186	140	543	.75	409	131	118	1611	328
16604	35	68	92	195	185	145	549	.78	430	126	128	1503	298
16603	42	61	86	189	186	140	550	.75	414	125	148	1551	293
16612	34	42	73	149	183	151	544	.83	449	129	110	1562	326
16611	26	38	68	132	185	147	545	.79	433	126	92	1550	343
16630	49	71	95	215	174	145	534	.83	445	128	124	1614	324
16631	38	72	97	207	186	143	543	.77	417	128	120	1630	330
16650	50	90	122	262	191	143	551	.75	413	131	116	1736	356
16651	46	82	101	229	191	141	552	.74	407	135	114	1701	351
16656	25	30	42	97	184	143	511	.78	397	122	112	1551	322
16657	28	37	43	108	186	143	535	.77	411	122	103	1556	332
16660	25	74	64	163	180	146	532	.81	432	130	122	1603	323
16661	41	78	65	184	179	144	527	.80	424	131	120	1579	320
16691	23	19	52	94	193	146	560	.76	424	134	119	1680	342
16690	23	36	59	118	191	145	551	.76	418	132	113	1696	351
16695	26	40	61	127	174	139	505	.80	403	125	86	1552	352
16694	16	37	50	103	174	138	500	.79	397	126	87	1548	349
16699	40	63	88	191	183	150	544	.82	446	128	115	1622	334
16698	40	54	90	184	192	147	556	.77	426	128	122	1624	327
16713	43	53	58	154	178	147	531	.83	439	130	114	1650	340
16712	48	60	85	193	177	150	534	.85	453	135	122	1659	334
16714*	44	69	95	208	186	137	532	.74	392	131	106	1695	358
16715*	34	86	106	226	185	140	536	.76	406	128	110	1679	350
16730	29	50	84	163	183	148	538	.81	435	128	100	1587	342
16731	43	69	96	208	186	146	541	.78	425	130	104	1627	346
16733	21	64	72	157	191	155	570	.81	463	142	143	1632	312
16732	33	59	63	155	183	158	565	.86	488	143	122	1606	324
16761	30	56	65	151	178	151	533	.85	452	130	97	1610	350
16762	26	51	75	152	176	154	540	.88	472	133	104	1598	340
16763	38	64	102	204	169	142	511	.84	429	121	88	1509	339
16764	31	48	95	174	174	145	523	.83	436	126	99	1530	331
16769	31	44	65	140	176	140	512	.80	407	125	102	1652	354
16770	33	31	60	124	177	138	508	.78	396	129	110	1676	350
16773	41	88	129	258	198	147	563	.74	418	133	138	1736	336
16774	52	85	130	267	194	147	560	.76	424	136	138	1743	337
16781	27	34	48	109	188	141	542	.75	406	120	111	1555	324
16782	38	55	46	139	189	138	537	.73	392	121	110	1563	326

* Caesarean

Monozygous males 1B

Number	M	R	V	T	L	B	C	I	E	Z	W	S	P
16557	45	47	56	148	175	159	534	.91	485	131	68	1486	364
16558	37	40	55	132	177	150	531	.85	450	127	66	1468	363
16574	40	30	48	118	177	145	531	.82	435	126	96	1450	317
16575	41	25	59	125	181	144	520	.80	414	129	112	1481	307
16582	48	55	80	183	195	148	551	.76	418	132	120	1735	352
16581	50	58	60	168	196	147	552	.75	414	132	118	1701	347
16583	42	51	72	165	190	150	555	.79	438	137	142	1792	343
16584	46	55	57	158	191	148	557	.77	432	131	132	1790	352
16593	—	43	64	107	183	151	524	.83	432	130	117	1661	340
16594	—	49	54	103	171	150	518	.88	454	132	116	1671	343
16597	45	51	83	179	187	135	516	.72	373	130	101	1624	349
16598	31	57	101	189	181	137	518	.76	392	123	99	1620	350
16602	44	60	83	187	195	152	562	.78	438	136	130	1725	341
16601	39	40	60	139	197	148	555	.75	417	134	126	1709	341
16626	40	43	46	129	186	156	560	.84	470	142	152	1699	318
16627	37	51	56	144	174	159	546	.91	499	141	133	1710	335
16628	44	66	81	147	203	154	581	.76	441	140	134	1693	330
16629	—	72	90	162	202	154	574	.76	338	140	141	1702	327
16633	37	37	46	120	180	148	530	.82	436	128	140	1795	346
16632	42	20	40	102	182	145	529	.80	421	129	141	1807	347
16635	27	36	37	100	185	148	536	.80	429	130	134	1723	337
16634	27	29	22	78	184	148	536	.80	431	129	120	1705	346
16653	45	72	98	215	186	160	547	.86	471	139	140	1767	340
16652	48	82	114	244	181	161	552	.89	491	139	143	1768	338
16658	51	46	128	225	193	157	566	.81	460	139	150	1715	323
16659	49	62	124	235	192	155	551	.81	445	139	138	1674	324
16681	44	47	92	183	192	140	542	.73	395	124	114	1663	343
16680	52	45	94	191	195	141	545	.72	394	123	122	1632	329
16684	44	64	64	172	186	149	542	.80	434	130	114	1600	330
16685	27	46	60	133	187	146	544	.78	425	130	115	1589	327
16688	29	49	38	116	193	150	556	.78	432	130	122	1709	345
16689	36	33	37	106	190	154	558	.81	452	136	140	1697	327
16692	27	20	39	86	192	147	549	.77	420	129	126	1748	349
16693	29	18	25	72	195	144	552	.74	408	130	134	1755	343
16696	48	62	71	181	199	153	573	.77	441	136	219	1704	283
16697	44	53	88	185	198	149	571	.75	430	135	220	1685	279
16708	33	30	86	149	199	145	555	.73	404	130	148	1714	324
16709	44	46	93	183	191	147	550	.77	423	131	134	1665	325
16718	39	54	51	144	190	156	554	.82	455	139	136	1735	337
16719	43	41	59	143	193	153	558	.79	442	141	136	1734	337
16720	47	73	86	206	191	151	560	.79	443	140	156	1752	325
16721	34	70	78	182	194	149	555	.77	426	140	162	1760	323
16746	54	63	92	209	208	154	586	.74	434	135	154	1680	313
16747	48	62	101	211	205	153	583	.75	435	136	145	1689	321
16756	32	39	93	164	191	148	554	.77	429	140	126	1752	349
16755	33	35	87	155	185	148	546	.80	437	139	124	1760	353

Dizygous females 1C

Number	M	R	V	T	L	B	C	I	E	Z	W	S	P
16553	48	85	73	206	185	146	543	.79	429	130	117	1645	336
16554	40	82	65	187	188	146	557	.78	433	135	130	1649	326
16555	39	69	119	227	197	147	560	.75	418	125	95	1656	363
16556	48	76	126	250	190	142	542	.75	405	127	90	1613	360
16560	50	75	115	240	179	151	541	.84	456	134	146	1587	301
16559	53	57	99	209	185	155	565	.84	473	139	138	1644	318
16588	44	62	59	165	183	141	532	.77	410	126	104	1632	347
16587	44	46	43	133	176	136	514	.77	397	124	96	1569	343
16589	36	31	65	132	169	135	491	.80	392	112	62	1391	351
16590	35	36	45	116	180	140	521	.78	405	120	86	1440	326
16605	35	68	87	190	190	145	547	.76	417	126	124	1656	332
16606	20	67	82	169	182	142	528	.78	412	128	116	1654	339
16624	42	75	116	191	191	138	560	.72	405	132	156	1690	313
16625	—	65	105	170	188	149	553	.79	438	132	138	1590	308
16637	50	88	110	248	185	144	548	.78	427	130	134	1671	327
16636	52	82	100	234	178	141	525	.79	416	125	121	1674	338
16676	23	72	85	157	182	135	539	.74	400	134	126	1700	338
16677	—	48	80	128	182	133	529	.73	387	129	115	1630	335
16679	41	65	119	184	174	146	522	.84	438	130	122	1557	313
16678	—	54	97	151	180	140	531	.78	413	110	147	1614	306
16683	37	47	69	153	172	145	525	.84	443	123	96	1594	348
16682	33	37	46	116	175	146	525	.83	438	126	106	1580	334
16687	32	65	93	190	180	145	529	.81	426	125	110	1642	343
16686	42	67	71	180	173	145	524	.84	439	125	108	1575	331
16716	29	61	85	175	179	142	529	.79	420	126	116	1589	326
16717	37	61	85	183	181	143	534	.79	422	129	103	1526	326
16725	21	32	60	113	179	140	514	.78	402	122	124	1605	322
16724	17	30	32	79	181	134	523	.74	387	120	98	1526	331
16726	17	72	90	179	183	141	532	.77	410	135	132	1650	324
16727	32	60	80	172	191	146	543	.76	415	132	114	1631	336
16735	26	25	55	106	192	144	555	.75	416	125	113	1587	328
16734	22	41	41	104	174	141	524	.81	425	127	121	1614	326
16736	28	31	26	85	193	145	562	.75	422	132	138	1635	316
16737	11	34	24	69	191	140	552	.73	405	126	136	1596	310
16742	47	47	67	161	183	151	548	.83	452	129	120	1672	339
16743	40	53	63	156	184	145	545	.79	429	134	148	1699	321
16767	46	68	96	210	187	142	536	.76	407	125	132	1675	329
16768	43	58	82	183	177	137	512	.77	396	123	131	1645	324
16771	30	51	49	130	183	143	536	.78	419	128	114	1601	330
16772	27	52	49	128	179	140	522	.78	408	128	110	1585	331
16775	34	58	65	157	177	146	518	.82	427	129	112	1591	330
16776	47	52	78	177	182	140	528	.77	406	129	94	1513	333
16784	11	41	61	113	190	153	550	.81	443	134	124	1640	329
16783	36	41	63	140	182	139	530	.76	405	123	92	1527	338
16778	42	77	68	187	186	147	553	.79	437	125	103	1541	329
16777	44	56	67	167	187	147	547	.79	430	125	94	1523	335

Dizygous males								1D					
	M	R	V	T	L	B	C	I	E	Z	W	S	P
Number													
16551	58	91	128	277	192	146	561	.76	427	127	136	1808	352
16552	54	73	129	256	195	144	561	.74	414	133	124	1683	338
16585	44	46	79	169	184	136	521	.74	385	120	101	1545	332
16586	42	34	42	118	189	136	534	.72	384	123	102	1626	348
16592	44	43	70	157	187	150	540	.80	433	127	110	1638	342
16591	43	36	58	137	187	151	541	.81	437	130	103	1588	339
16595	36	40	63	139	196	153	568	.78	443	132	144	1857	354
16596	42	39	63	144	186	159	553	.85	473	135	130	1691	334
16600	40	75	118	193	189	166	577	.88	507	142	155	1697	316
16599	—	57	78	135	190	155	565	.82	461	143	158	1684	311
16623	34	21	53	108	186	150	549	.81	443	132	122	1745	352
16622	45	31	70	146	194	147	559	.76	424	133	125	1650	330
16655	50	70	93	213	200	158	581	.79	459	135	132	1749	344
16654	45	67	109	221	198	156	575	.79	453	139	148	1760	333
16722	50	81	101	232	198	150	557	.76	422	129	107	1679	354
16723	41	47	96	184	195	144	555	.74	410	131	108	1695	356
16739	31	76	122	229	195	158	569	.81	461	139	134	1706	333
16738	43	70	75	188	199	148	564	.74	419	132	131	1706	336
16740	23	29	62	114	181	159	548	.88	481	139	132	1755	345
16741	26	29	42	97	190	152	556	.80	445	132	158	1730	320
16745	52	66	114	232	190	155	558	.82	455	139	134	1758	344
16744	42	69	120	231	182	159	554	.87	484	134	127	1745	347
16758	48	51	62	161	187	147	542	.79	426	134	126	1733	346
16757	30	35	49	114	186	154	550	.83	455	135	160	1705	314
16759	23	48	78	149	179	162	548	.91	496	140	123	1678	337
16760	38	62	87	187	187	165	563	.88	497	145	157	1680	311
16765	28	38	62	128	199	155	564	.78	439	146	153	1844	345
16766	55	70	105	230	204	154	580	.75	438	148	174	1818	326

differences between co-twins. Except when these differences are of zero magnitude their signs are either alike or different with regard to any two traits in a given pair of twins. With respect to two positively correlated traits the signs of the differences should more often be concordant than discordant; for two negatively correlated traits the signs of the differences should more frequently be discordant than concordant; and for two unrelated traits the signs should be discordant for about as many pairs of twins as they are concordant. The number of concordant and discordant pairs with reference to any two traits, X_i and X_j, is indicated by the notation $(X_iX_j, C:D)$, where C represents the number of concordant and D the number of discordant twin pairs. When it is clear by context which traits are being compared the notation is simply

C:D. The significance of the difference between the ratio C:D and the ratio 1:1 expected when there is no correlation betwen X_i and X_j can readily be tested by the binomial distribution. The value of C + D is commonly less than the total number of twin pairs, since co-twins are often tied for one or both of the measurements under consideration and therefore cannot be classified as either concordant or discordant with reference to them; besides, some measurements are missing from certain individuals. Table 2 gives the ratios C:D in monozygous females, monozygous males, dizygous females, and dizygous males for all combinations of traits taken two at a time except TM, TR, and TV. Also included in table 2 are the ratios C:D for monozygous males and females combined and for dizygous males and females combined.

RELATIONSHIP BETWEEN PSYCHOMETRIC TEST SCORES AND ANTHROPOMETRIC CHARACTERS

Table 2 affords some evidence that in monozygous twins the scores on the Verbal and Reasoning tests may be correlated with the size and shape of the head. Thus, for monozygous females we have (RL, 7:11), (VL, 6:12), (RB, 10:8), (VB, 10:8), (RI, 11:8), (VI, 12:7), (RE, 11:9), and (VE, 12:8). For monozygous males the corresponding ratios are (RL, 7:16), (VL, 8:15), (RB, 14:6), (VB, 15:5), (RI, 15:5), (VI, 13:7), (RE, 15:8), and (VE, 14:9). These ratios are consistent in direction between the two sexes and suggest that the scores on the Verbal and Reasoning tests in monozygous twins may be negatively correlated with Head Length and positively correlated with Head Breadth and Index of Cross-Sectional Head Area. But they are significantly different from 1:1 at the 5% level only for RI and VB in the males. There is little evidence for a sex difference in these ratios, and consequently it seems legitimate to pool the values of C:D for the two sexes. When this is done we have for the monozygous males and females combined (RL, 14:27), (VL, 14:27), (RB, 24:14), (VB, 25:13), (RI, 26:13) (VI, 25:14), (RE, 26:17), and (VE:26:17). These ratios again suggest that in monozygous twins both the Reasoning and Verbal tests may be negatively correlated with Head Length and positively correlated with Head Breadth, Cephalic Index, and Index of Cross-Sectional Head Area. However, none of these ratios for the combined sexes are significantly different from 1:1 at the 5% level.

The scores on the three psychological tests tend to be positively correlated with one another. It appeared likely, therefore, that some

TABLE 2

Ratios C : D of concordant to discordant twin pairs for all combinations of characters except TM, TR, and TV. See text for code of measurements

2A

Monozygous females above right
Monozygous males below left

	M	R	V	T	L	B	C	I	E	Z	W	S	P	O	
M		12 : 6	10 : 8	—	7 : 9	8 : 8	6 : 12	10 : 8	10 : 8	10 : 6	7 : 9	12 : 6	11 : 7	7 : 10	M
R	10 : 10		15 : 5	—	7 : 11	10 : 8	10 : 10	11 : 8	11 : 9	7 : 10	6 : 12	11 : 9	13 : 7	11 : 8	R
V	10 : 10	14 : 9		—	6 : 12	10 : 8	11 : 9	12 : 7	12 : 8	7 : 10	9 : 9	12 : 8	12 : 8	10 : 9	V
T	—	—	—		4 : 14	12 : 6	9 : 11	14 : 5	14 : 6	9 : 8	8 : 10	10 : 10	12 : 8	10 : 9	T
L	12 : 8	7 : 16	8 : 15	6 : 17		3 : 13	12 : 6	1 : 16	4 : 14	7 : 8	9 : 7	14 : 4	10 : 8	7 : 10	L
B	11 : 8	14 : 6	15 : 5	15 : 5	5 : 15		10 : 8	16 : 1	18 : 0	8 : 8	10 : 7	6 : 12	8 : 10	12 : 5	B
C	14 : 6	12 : 10	11 : 11	9 : 13	14 : 8	13 : 7		8 : 11	11 : 9	8 : 9	12 : 6	11 : 9	7 : 13	7 : 12	C
I	9 : 10	15 : 5	13 : 7	14 : 6	1 : 19	16 : 3	9 : 11		17 : 2	7 : 9	9 : 8	6 : 13	10 : 9	13 : 5	I
E	10 : 10	15 : 8	14 : 9	14 : 9	3 : 20	17 : 3	11 : 11	20 : 0		9 : 8	10 : 8	8 : 12	10 : 10	12 : 7	E
Z	11 : 4	8 : 9	9 : 8	8 : 9	9 : 8	6 : 9	8 : 8	9 : 7	9 : 8		9 : 6	9 : 8	6 : 11	3 : 13	Z
W	12 : 7	7 : 15	13 : 9	12 : 10	14 : 8	10 : 9	13 : 8	6 : 13	7 : 15	11 : 5		11 : 7	3 : 15	8 : 9	W
S	11 : 9	12 : 11	13 : 10	13 : 10	10 : 13	10 : 10	7 : 15	13 : 7	14 : 9	12 : 5	14 : 8		12 : 8	7 : 12	S
P	8 : 10	14 : 7	10 : 11	11 : 10	6 : 15	10 : 8	7 : 13	14 : 4	16 : 5	7 : 8	3 : 18	8 : 9		11 : 8	P
O	9 : 11	14 : 9	13 : 10	13 : 10	12 : 11	14 : 6	13 : 9	12 : 8	14 : 9	9 : 8	12 : 10	13 : 10	11 : 10		O
	M	R	V	T	L	B	C	I	E	Z	W	S	P	O	

2B

2B

Dizygous females above right
Dizygous males below left

	M	R	V	T	L	B	C	I	E	Z	W	S	P	O	
M		7:10	12:5	—	10:9	10:6	10:8	8:6	10:9	6:9	5:14	8:11	13:5	10:9	M
R	9:3		14:6	—	7:13	9:9	8:12	9:7	13:8	11:5	14:7	13:8	8:13	14:7	R
V	7:5	10:2		—	9:11	10:8	12:8	9:8	11:10	9:8	12:9	11:10	14:7	18:3	V
T	—	—	—		11:11	12:8	14:8	9:8	13:10	10:8	13:10	12:11	14:8	19:4	T
L	9:3	7:5	6:6	6:7		14:5	19:2	2:14	13:9	11:6	15:7	14:8	12:9	11:11	L
B	5:7	7:5	6:6	7:6	3:9		15:4	8:7	18:2	12:5	13:7	12:8	10:9	14:6	B
C	7:5	8:4	6:6	7:6	9:3	7:5		4:12	16:6	10:7	15:7	15:7	13:8	14:8	C
I	4:8	6:6	6:6	7:6	1:11	11:1	5:7		11:6	8:6	7:10	9:8	9:8	11:6	I
E	6:7	8:5	7:6	8:6	3:10	13:0	7:6	12:1		13:5	15:8	16:7	10:12	15:8	E
Z	5:8	4:9	7:6	8:6	7:6	6:7	8:5	5:8	6:8		13:5	12:6	7:10	9:9	Z
W	7:6	7:6	6:7	8:6	8:5	4:9	9:4	3:10	4:10	10:4		20:3	7:15	17:6	W
S	5:7	6:6	6:6	8:5	5:7	6:6	5:7	5:7	6:7	6:7	8:5		10:12	16:7	S
P	5:8	7:6	5:8	5:9	5:8	7:6	4:9	7:6	7:7	3:11	5:9	10:3		13:9	P
O	7:6	9:4	7:6	9:5	5:8	8:5	6:7	9:4	9:5	3:11	5:9	9:4	10:4		O
	M	R	V	T	L	B	C	I	E	Z	W	S	P	O	

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2C

Monozygous males and females above right
Dizygous males and females below left

	M	R	V	T	L	B	C	I	E	Z	W	S	P	O	
M		22: 16	20: 18	—	19: 17	19: 16	20: 18	19: 18	20: 18	21: 10	19: 16	23: 15	19: 17	16: 21	M
R	16: 13		29: 14	—	14: 27	24: 14	22: 20	26: 13	26: 17	15: 19	13: 27	23: 20	27: 14	25: 17	R
V	19: 10	24: 8		—	14: 27	25: 13	22: 20	25: 14	26: 17	16: 18	22: 18	25: 18	22: 19	23: 19	V
T	—	—	—		10: 31	27: 11	18: 24	28: 11	28: 15	17: 17	20: 20	23: 20	23: 18	23: 19	T
L	19: 12	14: 18	15: 17	17: 18		8: 28	26: 14	2: 35	7: 34	16: 16	23: 15	24: 17	16: 23	19: 21	L
B	15: 13	16: 14	16: 14	19: 14	17: 14		23: 15	32: 4	35: 3	14: 17	20: 16	16: 22	18: 18	26: 11	B
C	17: 13	16: 16	18: 14	21: 14	28: 5	22: 9		17: 22	22: 20	16: 17	25: 14	18: 24	14: 26	20: 21	C
I	12: 14	15: 13	15: 14	16: 14	3: 25	19: 8	9: 19		37: 2	16: 16	15: 21	19: 20	24: 13	25: 13	I
E	16: 16	21: 13	18: 16	21: 16	16: 19	31: 2	23: 12	23: 7		18: 16	17: 23	22: 21	26: 15	26: 16	E
Z	11: 17	15: 14	16: 14	18: 14	18: 12	18: 12	18: 12	13: 14	19: 13		20: 11	21: 13	13: 19	12: 21	Z
W	12: 20	21: 13	18: 16	21: 16	23: 12	17: 16	24: 11	10: 20	19: 18	23: 9		25: 15	6: 33	20: 19	W
S	13: 18	19: 14	17: 16	20: 16	19: 15	18: 14	20: 14	14: 15	22: 14	18: 13	28: 8		20: 17	20: 22	S
P	18: 13	15: 19	19: 15	19: 17	17: 17	17: 15	17: 17	16: 14	17: 19	10: 21	12: 24	20: 15		22: 18	P
O	17: 15	23: 11	25: 9	28: 9	16: 19	22: 11	20: 15	20: 10	24: 13	12: 20	22: 15	25: 11	23: 13		O
	M	R	V	T	L	B	C	I	E	Z	W	S	P	O	

function of these three measures would be more highly correlated with the anthropometric characters than would any one test alone. The function which was arbitrarily chosen is the sum of the scores on the three separate tests, which sum is called Total Test Score and is symbolized by T. The ratios C : D pertaining to the various anthropometric traits and Total Test Score are shown in table 2 also, and it is clear that these ratios confirm the relationships already suggested in monozygous twins between Head Length, Head Breadth, Cephalic Index, and Index of Cross-Sectional Head Area on the one hand and test scores on the other. In fact we have for monozygous males and females combined (TI, 28 : 11), (TL, 10 : 31), (TB, 27 : 11), and (TE, 28 : 15), of which the first two are significantly different from 1 : 1 at the 1% level, the third is significant at the 5% level, and the last is not quite significant at the 5% level by a two-tailed test. These ratios again suggest that in monozygous twins the psychometric tests considered in this paper tend to be positively correlated with Index of Cross-Sectional Head Area, Head Breadth, and Cephalic Index and negatively correlated with Head Length. These same relationships are present in the sample of dizygous twins also, but they are not significant at the 5% level. There is not much evidence in either monozygous or dizygous twins for relationships between test scores and any of the other anthropometric characters studied.

RELATIONSHIPS INVOLVING BIRTH ORDER

The relationship of each trait with Birth Order was investigated by subtracting the value of the trait in the second born co-twin from the corresponding value in the first born twin for each pair of twins in the sample. Except when a trait has the same value in both co-twins a pair of twins is either concordant or discordant with reference to the trait and Birth Order, according as the difference between co-twins in that trait is positive or negative.

The ratios C : D for the various traits and Birth Order, namely (OX_1, C : D) are given in table 2. There is some evidence that Stature is positively correlated with Birth Order in dizygous twins, the ratio (OS, 25 : 11) for dizygous males and females combined being significantly different from 1 : 1 at the 5% level. Moreover, there is some evidence for a positive relationship between head size and Birth Order both in monozygous and dizygous twins. Thus, for monozygous males and females combined we have (OB, 26 : 11), (OI, 25 : 13), and (OE, 26 : 16); for dizygous males and females combined the corresponding ratios are

(OB, 22:11), (OI, 20:10), and (OE, 24:13). Of these ratios only those for OB and OI in monozygous twins are significantly different from 1:1 at the 5% level, but the other ratios are consistent with one another and collectively suggest that there may be a tendency both in monozygous and dizygous twins for the co-twin with the broader head to be born first.

There is rather strong evidence for a relationship between scores on the psychological tests and Birth Order in dizygous twins. Thus, for dizygous males and females combined we have (OM, 17:15), (OR, 23:11), (OV, 25:9), and (OT, 28:9). The first two of these ratios are not significantly different from 1:1 at the 5% level, but the last two are significant at the 1% level. It appears likely, therefore, that in dizygous twins scores on the psychometric tests administered in this study are positively correlated with Birth Order. This relationship is not very evident in monozygous twins.

DISCUSSION

The data and analyses described herein appear to indicate that, particularly in monozygous twins, test scores may be correlated with head measurements, which in turn may be correlated with Birth Order. Moreover, there is evidence that in dizygous twins test scores and Stature may be correlated with Birth Order. It is possible, however, that these relationships are characteristic of our sample only and do not pertain to the population of twins from which the sample was taken. One of the strongest relationships observed in our sample is that between Total Test Score and Head Length in monozygous twins, but this ratio should be considered in the context of the 10 pairs of comparisons involving Total Test Score that were investigated, namely TO, TL, TB, TI, TC, TE, TZ, TW, TS, and TP. Each of these comparisons was made separately for monozygous and dizygous twins, making a total of 20 comparisons involving Total Test Score. In deciding whether the ratio (TL, 10:31) is really indicative of a relationship—in the population of monozygous twins represented by the sample—between Total Test Score and one or more of the 10 traits with which it was compared, it is necessary to consider—under the assumption that test scores are independent of all these characters—the probability of getting by chance in a set of 20 ratios at least one ratio that is as different from 1:1 as is 10:31. The most conservative way to compute this probability is to treat all 10 characters as independent—although in reality some of them are correlated with one another. The probability of drawing from

a population having the ratio 1:1 a particular random sample of 41 pairs with a ratio as asymmetric—or more so—than 10:31 is .00146, whence the probability of a particular sample having a ratio more symmetric than this is 1-.00146 = .99854. The probability of obtaining by chance at least one ratio as asymmetric as 10:31 out of 20 independent comparisons is therefore $1-(.99854)^{20} = .029$. In reality, three ratios involving Total Test Scores are significant at the 1% level, and one is significant at the 5% level. Moreover, the various characters are not all independent. The nine anthropometric characters were chosen without prior information concerning possible interrelationships with the three psychometric traits, and no other anthropometric characters were examined in this connection. Therefore, under the null hypothesis that Total Test Score is independent of all the anthropometric characters and of Birth Order, the value .029 may be regarded as an upper bound to the probability of obtaining at least one ratio involving Total Test Score as asymmetric, or more so, than 10:31. It seems reasonable to conclude that test scores are indeed a function of one or more of these characters, but the specific relationships observed by us require confirmation in other samples.

The observed relationships between mental test scores and head measurements suggest that a positive correlation may exist between test scores and general head size in both monozygous and dizygous twins. This conclusion is not immediately evident in monozygous twins, where Total Test Score is negatively correlated with both Head Length and Head Circumference. However, Total Test Score is positively correlated with Head Breadth and with Index of Cross-Sectional Head Area in monozygous as well as in dizygous twins. The negative correlation of Total Test Score with Head Length and Head Circumference in monozygous twins may result from the strong negative correlation in monozygous twins between Head Length and Head Breadth, which relationship may well be responsible for Index of Cross-Sectional Head Area being negatively correlated with Head Length and essentially uncorrelated with Head Circumference.

The conclusion that mental test scores are positively correlated with head size in like-sexed twins is in agreement with the finding of Pearson (1902, 1906, 1925) and Pearl (1906) for the general non-twin population, except that in twins—particularly monozygous twins—the correlations are very much stronger than in the general population. A possible interpretation of these results would seem to be that test scores

are a function of both brain quality and brain quantity. In monozygous twins qualitative differences between co-twins are probably small, so that differences in test scores are mostly a function of differences in brain size. In dizygous twins qualitative differences between co-twins are greater than in monozygous twins, and differences between co-twins in test scores are less highly correlated with differences in brain size than in the case of monozygous twins. In the general population, however, qualitative differences between individuals are realtively very large, as a consequence of which there is only a very weak correlation between brain size and test scores.

The relationship between head size and mental test scores observed by us in twins and by Pearson and by Pearl in non-twins has some interesting implications regarding a possible relationship between intelligence and fertility. Clark and Spuhler (1959) have observed a positive correlation between fertility and head size in males in a random sample of adults from the population of Ann Arbor, Michigan. This observation, combined with those of the present paper, suggests that in males fertility may be positively correlated with intelligence in the Ann Arbor population.

The relationship between Birth Order and test scores in dizygous twins cannot be explained by the data available in this investigation. If it were true, however, that an individual takes more than a passive part in its own birth it would be possible that the more vigorous twin would usually be born first. And if vigor at birth were positively correlated with test scores at adolescence the relationship observed between Birth Order and test scores would be expected. On the other hand, it is possible that in dizygous pairs both the Birth Order and the test scores of a twin are functions of its position in the uterus, the individual that is located in such a way as to be born first also having a broader head, greater stature, and higher test scores than its co-twin. It is interesting that Head Breadth appears to be related to Birth Order in both monozygous and dizygous twins, whereas test scores and Stature are related to birth order only in dizygous pairs.

If scores on certain psychometric tests tend to be rather highly correlated with brain size in monozygous twins and with birth order in dizygous twins, as is apparently the case, this would seem to provide an additional criterion for comparing the validity of psychometric tests as measures of innate mental ability. Tests which in twins are rather highly correlated with head measurements and Birth Order may be better

indicators of innate capacity than those which are only slightly correlated with these characters. By this criterion the Chicago Tests of Verbal and Reasoning Ability are better measures of innate mental capacity than is the Progressive Matrices Test. It should be noted that this conclusion is at variance with earlier views regarding the relative merits of these tests. For it has generally been assumed that non-verbal tests, like the Progressive Matrices Test, inasmuch as they measure skills not commonly taught in school, are relatively "culture free" and hence are better measures of innate capacity than are tests which involve skills that are usually taught in school. More recent findings, however, are in agreement with our own results in suggesting that these assumptions should be reexamined. Thus Higgins and Sivers (1958) have observed that culturally handicapped Negro children score relatively higher on verbal than on non-verbal tests, and Walters (1958) has come to the same conclusion regarding Maori children in New Zealand.

SUMMARY

The interrelationships among certain psychometric and anthropometric characters and Birth Order were investigated within pairs of like-sexed twins. The relationship between any two traits, X_i and X_j, was investigated by comparing the signed difference between co-twins in trait X_i with the corresponding difference in trait X_j for each pair of twins. Two traits are positively correlated, negatively correlated, or uncorrelated in a given sample of twins according as the signs of the differences between co-twins in the two traits are alike in more twin pairs than they are different, different in more pairs than they are alike, or alike in about the same number of pairs as they are different.

A positive correlation was found between scores on certain psychometric tests and head size in both monozygous and like-sexed dizygous twins. The relationship is significant at the 5% level in monozygous twins but is weaker and not significant at the 5% level in dizygous twins. The same relationship was found by Pearson and by Pearl to obtain with much lower intensity in the general non-twin population. Combined with the finding by Clark and Spuhler that fertility is positively correlated with head size in the male population of Ann Arbor these observations suggest the possibility that fertility may be positively correlated with intelligence in this population.

In dizygous twins there is a positive correlation, significant at the

1% level, between scores on the psychomteric tests and Birth Order, the first born co-twin usually scoring higher than the second born twin.

The existence of correlations between scores on certain psychometric tests and head measurements and Birth Order suggests an additional criterion for comparing the efficiency of various psychological tests as measures of innate ability. Tests which correlate highly with these physical measurements may be better measures of innate capacity than those which do not.

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THE AGE AT MENARCHE IN SOUTH AFRICAN BANTU (NEGRO) GIRLS:

WITH A COMMENT ON METHODS OF DETERMINING MEAN AGE AT MENARCHE

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IN the Bantu-speaking Negroes of South Africa the incidence of cancer of the female genital organs differs significantly from that seen in more westernized populations. Carcinoma of the female breast, ovary and endometrium are strikingly less common than in United States whites or Negroes, while carcinoma of the cervix is more frequent than in United States whites, and aproaches the incidence reported for United States Negroes (Oettlé and Higginson, 1957). Since these observed differences may reflect endocrine alterations attributable to environmental influences, certain aspects of endocrine activity were examined, including an assessment of mean age at menarche.

Racial differences in the mean age of the menarche have long been recognized, and are increasingly believed to be environmental in origin. The group we investigated was composed of urbanized Bantu girls of Alexandra Township adjoining Johannesburg.

METHOD

The information was obtained during a routine diphtheria immunization program among schoolchildren. Schools were taken in their order on the immunization program, and were visited between October 4, 1955 and April 13, 1956. They were unselected except as regards the ages of the scholars.

As each schoolgirl received her injection, she was asked her date of birth and whether menstruation had occurred. It was soon found that privacy was desirable, and once this was ensured, refusals became very infrequent. Names were not recorded, so a further source of possible reluctance was removed.

One thousand and thirteen girls were interrogated. All knew the year of birth, but eleven refused to give any information regarding their periods. These girls do not differ from the remainder as regards knowledge of the month of birth (table 1), and their omission does not alter the results significantly.

TABLE 1

Age of menarche in Alexandra Township schoolgirls according to age

		GIVING INFORMATION				REFUSALS	
AGE LAST BIRTHDAY	TOTAL	NUMBER ATTAINED MENARCHE	PERCENTAGE OF AGE GROUP	NUMBER KNOWING MONTH OF BIRTH	PERCENTAGE OF AGE GROUP	NUMBER REFUSING TO ANSWER REGARDING MENARCHE	NUMBER KNOWING MONTH OF BIRTH
9	19	0	0	2	10.5	0	0
10	90	1	1.11	42	46.7	0	0
11	139	3	2.16	65	46.8	3	2
12	155	14	9.03	118	76.1	1	1
13	198	33	16.67	170	85.9	4	1
14	143	60	41.96	134	93.7	2	1
15	109	69	63.30	102	93.6	1	1
16	69	60	86.96	68	98.6	0	0
17	39	38	97.44	39	100.0	0	0
18	21	20	95.24	21	100.0	0	0
19	15	15	100.00	15	100.0	0	0
20	5	5	100.00	5	100.0	0	0
Total	1002	318	31.74	781	77.9	11	6
							(8.4 expected)

Of the remaining 1002, 781 knew the month of birth, many claiming to know the day. The distribution of birth months does not differ significantly from random (table 2), so there is no evidence of bias in the ages stated. As regards the 221 girls in whom the birth month was not known, it seemed legitimate to allocate these systematically to successive months of the year, and from this arbitrary date, to calculate age at last birthday on the date of interview. These were all younger girls by comparison with those who knew their birthdates, and included only twelve in whom menses had commenced. Separate analysis showed that slightly fewer of those ignorant of their month of birth had attained

maturity than would have been expected, but this difference was not significant ($p = 0.4$), so the stated year of birth in these girls could be accepted as reliable.

RESULTS

The figures obtained on the 1002 girls vouchsafing information are given in table 1, together with information on the refusals. By probit analysis the mean age at menarche is 14.89 years, with standard deviation 1.66, standard error 0.093 years, and standard deviation of (b) — 0.0107.

Probit analyses assumes a normal distribution of the population, from which the different age samples have been drawn, but calculates

TABLE 2

Birthmonths of 781 girls willing to give information on age at menarche

	OBSERVED	EXPECTED
January	61	66.3
February	51	59.9
March	58	66.3
April	84	64.2
May	73	66.3
June	75	64.2
July	69	66.3
August	63	66.3
September	65	64.2
October	54	66.3
November	67	64.2
December	61	66.3
	781	780.8

$\chi^2 = 14.553$ with 11 d. f., $p = 0.20$

the sigmoid accumulative frequency curve from the values of these age samples, and transforms this curve into a straight line by means of probits. The mean of this distribution corresponds to age at which 50 percent of the girls have attained menarche in the present population not in the sample. Probit analysis thus avoids the distortion introduced by selection of age groups and estimates the population mean. The standard deviation given is the standard deviation of the present normal distribution, and is the reciprocal of the slope of the probit line.

The empirical probits cluster about a reasonably straight line, *i. e.*, there is no demonstrable tendency for these girls to leave school on

attaining physiological maturity. This conclusion was in agreement with the impressions of Bantu social workers at Entokosweni. A very different situation apparently exists among Natal Indian girls, where the menarche tends to precipitate school leaving (Kark, 1953).

DISCUSSION

Methods of investigating mean age at menarche.

The present system of analysis in which only two facts are required, *viz.*, age when interrogated and the *status quo* as regards commencement of the menses, with subsequent probit or logit analysis, is the simplest method of collecting information of high validity. Hogben, Waterhouse and Hogben (1948), made use of both probit and logit analysis, and preferred the latter on theoretical grounds, since probit analysis arbitrarily assumes a normal distribution of the proportion of people of an age group attaining a given level of maturity. They did not, however, use the first menstruation as the critical date, so the mean age calculated by these workers is somewhat later than that found by Wilson and Sutherland (1950a and b) and cannot be compared with the results of others. Probit analysis is the method chosen by Wilson and Sutherland (1950a) for data of this type, and provides a very satisfactory approximation to a straight line, which suggests that the assumption of a normal distribution is realistic.

Other methods, by comparison, are either immensely tedious or subject to uncontrolled inaccuracies. Best of these are prospective surveys, such as that of Engle and Shelesnyak (1934), who used trained personnel to keep careful records of each girl from the time of admission to an institution until the date of her menarche.

A second type of survey, of which Michelson's (1944) study is an example, employes retrospective statements. Since this introduces the factor of poor memory, Michelson rejected all who could not remember the month during which the first menstruation occurred, and as for those who could remember the month, reasons had to be given why this date was remembered. Of about 7,000 individuals questioned, only half could provide the satisfactory information. His method of confirmation does not exclude errors of memory, which moreover may not be random. Furthermore, such errors may vary according to the cultural and educational background of the population. Both Kark (1943) and Ellis (1950) have noted a tendency for older girls to state an older age at menarche. Retrospective studies only measure a past situation and

cannot, however accurate, be regarded as relating to the date of the survey. Thus Benjamin (1960) found a mean age at menarche of 14.6 years for 1,444 postmenopausal white women of the Cape Province attending a hospital in Cape Town. His results relate to the South African situation at least thirty-five years ago—and for his older patients even earlier than this. In addition, his survey demonstrates errors of memory for recent events in the form of prominent peaks at the ages of 40 and 50 in the recollected age of menopause.

TABLE 3

Attainment of menarche compared with knowledge of birthmonth

AGE AT LAST BIRTHDAY	NUMBER KNOWING MONTH	NUMBER ATTAINED MENARCHE	NUMBER NOT KNOWING MONTH	NUMBER ATTAINED MENARCHE
9	2	0	17	0
10	42	1	48	0
11	65	1	74	2
12	118	12	37	2
13	170	31	28	2
14	134	57	9	3
15	102	66	7	3
16	68	60	1	0
17	39	38	—	—
18	21	20	—	—
19	15	15	—	—
20	5	5	—	—
Total	781	306	221	12

Another weakness of the retrospective method is that it discards those persons whose memories are patently defective, yet no evidence is usually available to show that those with poor memories are truly comparable with those whose memories, for one reason or another, are more accurate. The omission of a section of the population thus introduces yet another possible source of bias.

It will be evident that both prospective and retrospective studies are susceptible to bias. A prospective study requires a closed population in which every member is prepubertal on admission and is followed until every member has attained the menarche. This could not be achieved in Kark's (1956) study of Indian girls, in which the initial survey of 1259 girls contained 748 who had not attained the menarche. A follow-

up was possible on 528 (70.6%) of whom 392 attained the menarche (74.2% of those followed). Her calculations were necessarily limited to the latter.

Retrospective studies, as distinct from prospective and *status quo* methods, should be undertaken only when the entire population has attained the menarche before interview, otherwise the younger the group investigated, the lower will be the figure obtained for its average age at menstruation. This is well illustrated by Michelson's (1944) figures and those of Wilson and Sutherland (1949). The latter found the average age of menarche for Oxford city schools to be 13.40 years, and for Oxfordshire country schools, 13.09 years. That this was solely due to the younger age of country schoolgirls was shown by probit analysis, from which the mean age of 13.6 was calculated for both groups.

Comparison with other Bantu groups.

Only one other study of age at menarche in Bantu girls is available. Kark (1943) analyzed figures collected in the main from rural districts in the Northern Transvaal (562 girls), and in the Cape Province and South Natal (476 girls). Ages ranged from 6 to 25 years, 361 belonging to age groups in which none had commenced to menstruate, 105 were postpubertal, and only 572 were of value in the determination of mean age at menarche. There is some likelihood that the ages given by the girls were incorrect, because registration of births did not occur; she states that where the given age was obviously incorrect an approximate age was recorded. This estimate might easily be influenced by the presence or absence of the anatomical changes associated with puberty. Kark regarded the stated age of onset of menstruation as unreliable, since girls of 18 to 20 gave 15 to 16 years as their age of onset of menses, whereas those of 15 to 17 gave an age of 13 to 14 years for their age at menarche. She therefore discarded statements based on memory and recorded simply the percentage in each age group in whom menses had commenced. For the northern areas she found that the figures were approximately one year later than those found for the southern areas.

Crude analysis of her figures give a mean age at menarche for the northern areas of about 16 years, while that for the southern areas is about 15 years. While the southern areas give results not very different from ours, she was supplied with no record of a girl under 13 years of age in whom menstruation had commenced, whereas 9% of our girls aged 12 had reached menarche.

The northern areas were hotter than the southern, with a much higher incidence of chronic malaria, bilharziasis, and malnutrition.

Comparison with other populations.

The mean age obtained in our Alexandra township Bantu schoolgirls is distinctly older (table 4) than those in schoolgirls in England or Nigerian Negroes (Ellis, 1950, Wilson and Sutherland, 1953), or in Indian girls in Durban (Kark, 1956), or Negroes in New York (Michelson, 1944). They are closer to the figures abtained for pagan Negroes in northern Nigeria, aboriginals in Central India or rural Cingalese (Wilson and Sutherland, 1950a, 1953), although our mean figure is older than all of these.

Significance of mean age at menarche.

Many views have been put forward to explain the menarchal variation between groups differing in race, habitation, or both. Much of the older work is speculative and based on methods of dubious validity. There seems little point in considering it in detail.

It would be unwise to ignore genetic factors, particularly as regards individual cases. Mills (1937) held that climate was also important, concluding that "adequacy of diet cannot overcome the physical retardation that comes with difficulty of body heat loss" (page 56). He based this view on the later onset of menstruation in tropical regions, but tropical diseases were then poorly controlled, and nowadays few would consider that the effect of temperature on menarche merits serious consideration.

As regards population as a whole, there is evidence in support of the view that nutrition is somehow important. Keys *et al.* (1950) have summarized the many reports indicating that the onset of menstruation is delayed by periods of severe food shortage. Where social classes can be distinguished within a racially homogeneous population, those with a poorer nutritional status show a later mean age at menarche. For example, Bojlén, Rasch, and Bentzon found a difference between Danish girls from economically straightened families (13.80 years) and those of professional parents (13.65 years). Kark (1956) showed for Durban Indian girls from social classes 1, 2, and 3, a mean age of 13.44, and from social classes 4 and 5 a mean age of 13.71 years. Michelson (1944) has gone so far as to state that when social classes are equal, so are the mean ages at menarche. However, Wilson and Sutherland (1950b)

TABLE 4

Average age of the menarche

RACE	AREA	NO. OF GIRLS	MEAN	S.D.	S.E.	REFERENCE
Bantu	Alexandra Township	1,002	14.89	1.66	±0.093	This study
Negro	North Nigeria plateau pagans	74	14.50	1.24	—	Wilson and Sutherland (1953)
Negro	North Nigeria School	172	14.14	0.61	—	Wilson and Sutherland (1953)
Negro, mainly Ibo and Yoruba	Southern Nigeria Series 1	300	14.22	1.00	—	Ellis (1950)
	Series 2	142	14.40	—	—	Ellis (1950)
Indian	Durban	528	13.60	1.13	±0.06	Kark (1956)
Indian aboriginals	Central India	411	14.65	1.55	—	Wilson and Sutherland (1953)
Cingalese	Rural	296	14.39	1.73	±0.16	Wilson and Sutherland (1950a)
Cingalese, Whites, Tamils	Colombo City Schools	844	12.84	1.24	±0.07	Wilson and Sutherland (1950a)
American Negro	New York	1,397	13.40	—	—	Michelson (1944)
White	New York	1,922	13.08	—	—	Michelson (1944)
White	South England	2,590	13.49	1.19	±0.037	Wilson and Sutherland (1950b)
White	Copenhagen	17,557	13.8	1.0	—	Bojlén *et al.* (1954)

The mean, standard deviation and standard error given by authors in the above table have been derived by probit analysis in the present study, and in the publications of Wilson and Sutherland, Bojlén *et al.*, and apparently, of Ellis.

found the mean age at a Sussex boarding school to be 13.11, while that at a Yorkshire school was 13.82 years—a difference that could not be explained simply on social class difference. Higginson and Oettlé (1960) have previously reported on the nutritional and socioeconomic background of the present group. These studies indicated that the income in 52% of households in this township in 1953 was below the "poverty datum" line, *i. e.*, below a theoretical estimate of income needed by any household if it is to obtain a minimum of health and decency. The nutritional studies showed the average diet was low in fat and animal protein, and that the nutritional status of the population as a whole was below that of European or United States groups.

Our results are consistent with the hypothesis that the difference between Bantu schoolgirls and their United States Negro counterparts could be most simply explained on a basis of poorer nutritional status of the Bantu, difficult as this may be to define. However, the relationship to nutrition is far from complete, and further studies are clearly indicated to clarify the significance of the age at menarche as a measure of endocrine activity in relation to specific nutrient and dietary deficiencies, before ascribing these results to nutritional deficiency alone.

CONCLUSIONS

1. The mean age of menarch in 1002 Alexandra schoolgirls near Johannesburg was found to be 14.89 years. This is somewhat older than is generally observed and it is suggested that this may be an expression of the poorer nutritional and socioeconomic status of this population.
2. Probit analysis is recommended for such investigations.

ACKNOWLEDGEMENTS

We are indebted to Sister Hilda Madikiza of the Entokosweni family welfare center in Alexandra township for collecting the information. The schools visited were: 4th Avenue, 10th Avenue, Dorky, Entokosweni, Africa, Pholoshoko, Emfundisweni Community, Holy Cross, St. Michael's and the 3rd Avenue.

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BOOK REVIEWS

Human Growth. J. M. TANNER (ed.) vii + 120 pp. Pergamon Press, New York, London. 1960. $5.00.

The study of human growth lapsed and languished in Europe until the 1950's when a number of growth centers were established, and when numerous symposia and meetings were devoted to growth in its various parts.

The present volume is the result of one of the more recent symposia. It is modest both in scope and size. It contains essays by Adolph Schultz (age changes in primates), methods of analysis of human growth (Wilma J. Israelson), a brief section on human growth genetics (by J. M. Tanner), a chapter on race and climatic effects (by D. F. Roberts), material by Roy Acheson on the effects of nutrition and disease, a three-page note on prenatal influences, and finally, a longish review of secular changes in stature and "intelligence" (by A. W. Boyne).

Professor Schultz's contribution, as usual, stands on a summit by itself and provides clear documentation of the remarkable skeletal immaturity of the human primate at birth. The bulk of the papers, however, are not so novel in content; in fact they have a curiously dated flavor. They are well-done, to be sure, but at the same time detached from contemporary problems of biological import. Thus, Dr. Israelson's summary of methods of analysis of growth is exemplary of itself, but it never comes to grips with the reasons for such growth analyses. Similarly, Boyne's paper in the well-grooved area of secular changes does not grapple with factors affecting the tempo of maturity and the rate of growth. Even D. F. Roberts' summary of race and climate (which contains some interesting information on the apparent lack of effect of the sickling gene on growth) is primarily descriptive rather than investigative.

Acheson's paper on nutrition and disease, therefore, comes nearest to connecting this volume with experimental biology and pediatric endocrinology. Acheson concerns himself with the reasons why growth may be altered under particular circumstances of caloric deprivation or protein lack (even though he intentionally avoids data on kwashiokor and marasmus). Acheson's bones are not osteological specimens, but living tissues provided with collagen, phosphatase and mucopolysaccharides.

His bibliography, too, will be useful to many for it is a biological and medical bibliography.

Without question this book will be valuable for teaching and valuable for the teacher. At the same time it illustrates to perfection how "growth" became an intellectual isolate with the few growth specialists talking growth to each other. Actually this volume also begins to point the long voyage back with growth viewed in context of the growing organism.

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Heights and Weights of Adults in the U. S. By M. L. HATHAWAY and E. D. FOARD, Home Economics Research Reports No. 10. Human Nutrition Research Div., Agric. Research Service, U. S. Dept. Agric. Washington D. C., August. 1960. Pp. vi and 110; 70 Tables; 35 Figures; bibliography. (U. S. Printing Office, Supt. Documents, 60¢)

This is a companion monograph to Hathaway's 1957 "Heights and Weights of Children and Youth in the U. S.," which is Research Report No. 2. The same format is followed, and there is the same evident care in the selection and tabulation of data. If anything, I'd say the present monograph is broader in scope. Further, I find frequent graphic summaries of tabular data a great help.

The period covered by the study is roughly 1850-1955 and is based on both published and unpublished data. The reporting is in terms of four U. S. geographic areas: North East, North Central, South, and West. There are five major Sections: average heights and weights related to age; average weights for height and age; data on males in military service; comparative U. S.—other country data (Canada, Great Britain, Sweden, Norway, Denmark); U. S. population changes 1790-1950.

Certain interesting data on height and weight emerge: for males stature is taller in order of NE or NC → South → West (even though Texas is listed under South!); females are taller in the West; male height was less in rural areas earlier, now is greater; male weight is

less or equal in rural areas; males are taller and heavier if the homemaker is college-bred; female height is taller, but weight is less, if the homemaker is college-bred.

Familial data on stature are quite illuminating: r for daughter-father height is 0.49, for daughter-mother height is 0.51; $Y = 32.98 + 0.498X$ and $Y = 35.21 + 0.427X$, where Y = height of daughter, X = height of father or mother, respectively. Each r differed significantly from zero at the 1% level, and when squared was ca. 0.25. Hence, 25% of variance of daughters' height is due to the linear regression of Y or X, and 75% due to factors other than each parent's stature.

Compared with U. S. whites American Negroes are heavier for height in both sexes, markedly so in the Negro female. For height U. S. males are tallest, then Norway, then Canada; Norway females are tallest to 35 years, then U. S. females take over, with Canada shortest. For weight U. S. males are heaviest, then Canada, then Norway; Norway females are heaviest to 45 years, then U. S. females catch up, although at 60 years the three are about equal.

The population data do not differ in any significant detail from those in many current publications and recent U. S. and U. N. releases.

In a final evaluation the authors ascribe U. S. height increase over the years to: increase in growth potential; improved socio-economic status; better medical care and sanitation; availability of more nutritious foods plus better knowledge of nutritive needs.

This is a well documented, useful monograph. You'll not get more for your 60¢ in a long, long time!

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Handbook of Aging and the Individual: Psychological and Biological Aspects. JAMES E. BIRREN (ed.). xii + 939 pp. The University of Chicago Press, 1959. $12.50.

The Biology of Aging: A Symposium. BERNARD L. STREHLER (ed.), with James D. Ebert, H. Bentley Glass, and Nathan W. Shock. vii + 364 pp. American Institute of Biological Sciences, Washington, D. C., Publ. No. 6, 1960. $7.75.

Aging: Some Social and Biological Aspects. NATHAN W. SHOCK (ed.). viii + 427 pp. American Association for the Advancement of Science, Washington, D. C., Publ. No. 65, 1960. $8.50.

Aging is a universal biological phenomenon—a fact demonstrated with inexorable certitude by all the biological clocks, wound up at conception, coming eventually to a stop. Like human growth and development or starvation, aging is manifested in a vast array of changes, at all levels of organization: cells and tissues, single organs and organ systems, total organism considered individually and in its interaction with the social milieu. Aging is one of the focal concepts and concerns of human biology.

In recent months it was as if a featherbed filled with publications on gerontology were torn wide open, and the stuffing of studies of aging viewed as a complex biosocial phenomenon spewed forth. "Biology" or "Biological" appears in the title or the subtitle of the three books under review. None of them is concerned strictly or solely with human biology but all are relevant to it.

In part, the sudden upsurge of interest in the biology of aging is an artifact resulting from the delay in the processing of one of the symposia. But the general increase of research activity in this field reflects increased federal support and stimulation of investigative work, and the growing significance of gerontology and geriatrics, the medical specialty concerned with old age.

The first volume, now in its second printing, is a systematic treatise focused on age changes in the various aspects of behavior of individuals. While "biology" in the subtitle of the volume could be largely supplanted by "neurobiology," some chapters can be safely labelled "general biology" and "human biology." A chapter is devoted to the treatment of problems of aging in terms of mathematical models. The role of genetic concepts and mechanisms in interpreting individual variations

in the patterns of aging is discussed, with brief reference to systems of body-build description. Bodily changes with age are considered in some detail (pp. 401-408) and there is a chapter on health, morbidity and mortality.

The aim of the AAAS symposium was two fold: to review briefly the current status of research on various aspects of aging and to identify fruitful, urgent problem areas for investigation. We are informed in the preface that the participants were asked to place primary emphasis on the development of ideas rather than on detailed reviews of literature. Thus the volume does not replace the need for a new edition of *Cowdry's Problems of Aging: Biological and Medical Aspects*, the third (1952) edition of which was prepared by A. I. Lansing.

Human biologists will find some materials that are of direct interest in all five sections of the proceedings, entitled Implications for Society (demographic factors, biological aspects of senescence); Aging in the Tissues and Cells (genetics, structural changes); Aging in the Total Organism (radiation, cardiovascular system, steroid hormones); Theories of Aging; and for good measure, Oral Aspects of Aging (structural age changes in human teeth).

In this reviewer's opinion Section IV (Theories of Aging) is the core of the symposium. At the outset, N. W. Shock marshall's an array of diverse facts which must be taken into account by theorists of aging. Since this is at the same time a succinct summary of gerontology's major findings, we shall list them:

(1) The probability of death increases logarithmically with age, whereas measurements of functional capacities decline linearly with time; (2) longevity is related to genetic characteristics; (3) males of most species show shorter life spans than females; (4) life span can be influenced by changes in diet; (5) in poikilothermal animals, life span is shortened by increasing the environmental temperature and increased by lowering the temperature; (6) exposure to nonlethal doses of radiation shortens life span; (7) the rate of change with age differs among different organ systems; (8) with increasing age, there is a reduction in reserve capacities as indicated by the reaction of the individual (or organ system) to stress; (9) age changes are greater in total animal (or organ) performance than in intracellular biochemical processes; and (10) age changes increase with the complexity of the performance measured.

In Shock's opinion the impairments of performance in man, associated with advancing age, are due to (a) dropping out of functional

units in key organ systems, (b) some impairments in the functional capacities of cells remaining in the body of the aged, and (c) breakdown of neural and endocrine integrative functions in the individual.

H. Selye, with P. Prioreschi, surveys the relationship between aging and the "adaptation syndromes," local and general, which develop during "stress." B. L. Strehler's point of departure is the idea that living systems are essentially a special type of energy transducers. Aging is defined in this context as a predictable failure of these transducers, resulting from the inadequacy of their repair. J. E. Birren reviews and enriches theories which attempt to account for behavioral changes taking place with advancing age. S. E. Stumf contributes a philosopher's views of aging.

The volume edited by Strehler is outspokenly "fundamental" in its orientation. The leading question is: What are the basic mechanisms underlying the gross phenomenon of aging? Or put in another way (N. W. Shock): Why does the probability of death increase with age? Some of the attempts to answer these questions are not irrelevant for human biologists. The problem is approached—and this technique is of general interest—in 2 ways: 1. In the form of 50 "ideas and results" papers supplied by the participants to their colleagues prior to the meetings, and 2. a revised and edited text of discussions on a variety of topics, including mortality rates and genetic aspects of aging.

The fact that chronic exposure of laboratory animals to low-dosage ionizing radiation accelerates the aging process is of interest from the point of view of radiobiology. At the same time, it may shed light on the mechanism of "normal aging." Radiation effects are histopathologically nonspecific. G. W. Casarett (p. 112) concludes that irradiation advances the "physiological debility" of an aging organism, bringing about an earlier onset of the diseases of old age and thus shortening life.

Along more "curricular" lines is S. M. Garn's contribution on fat accumulation and aging in males and females. While questioning whether fattening is an inevitable concomitance of age, Garn documents teleroentgenogrammetrically the old and concise adage that "men run to belly and women to bum." Data on median subcutaneous fat thicknesses in Ohio subjects, age 20 to 40 and 40 years plus, respectively, indicate that in the older group the fat layer increased at all sites that were measured, with the largest increments at the iliac crest in the males and at the mid-trochanteric site in the females.

Reference to body-composition studies based on the determination of the total and extracellular body water was made by N. W. Shock.

With intracellular water considered as an index of the total amount of protoplasm and a measure of the "metabolic size" of the human organism, the age decrement in basal metabolism (calculated usually as oxygen intake per unit of surface area) disappears, *i. e.*, it is accounted for by the loss of protoplasm with increasing age (plus, let us add, an increase in the fat content of the body). Evidence of tissue loss with increasing age was brought out in studies on rat muscle. There was a reduction of about 30% in the net weight of the muscle of the hind leg in old male rats as compared with young adults. In addition, there was a small decrease in protoplasm per unit of remaining tissue. Shock concludes that loss of protoplasm is an important factor in the age decrement in the performance capacity of the total animal.

A point of signal importance for human biology is raised by the repeatedly verified observation, reported by C. M. McCay and his coworkers for aging rats (1943 Arch. Biochem. 2:469-479) and further documented at the symposium by B. N. Berg, that the onset of diseases of old age is significantly delayed by retarding the rats' growth and freedom from adult obesity. Restriction to levels of 33% (moderate restriction) and 46% (severe restriction) below the *ad libitum* level of food intake, from weaning to 800 days of age, resulted in 5% to 7% reduction in tibia length, 6% to 13% reduction in body length, and 25% to 40% reduction of body weight, the latter difference being largely due to the absence of excess body fat. Female mortality was low in both groups (5% at 800 days) and unaffected by the dietary restriction. In males the mortality was 15% in the restricted rats, 50 (fifty)% in the unrestricted rats. Lesions indicative of myocardial degeneration were found in all (100%) of the unrestricted, in 64% of the moderately restricted, and only in 24% of the severely restricted males. For females the corresponding figures were 60%, 0%, and 0%, i. e., the restricted females were free from lesions. Maximum rate of growth and large body weight, indicative of fatness, are inimical to longevity; they impair female fertility, regarded as criterion of the organism's vitality.

The findings were cited here in some detail since the problem has far-reaching potential implications for man. If human biologists get nothing more out of the book than the realization of the urgency of studying this problem, the investment of $7.75 would be well worth it.

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BOOK NOTICES

HANDBOOK OF CIRCULATION. Analyzed and compiled by Philip L. Altman. Edited by Dorothy S. Dittmer and Rudolph M. Grebe. x + 393 pp. Saunders, Philadelphia, 1959. No price indicated. The explosive growth of information in biology, largely quantitative in nature, makes synthesizing efforts a prerequisite of intellectual survival or, at least, of effective utilization of scientific information. This is the fifth "Handbook" prepared under the direction of the Committee on the Handbook of Biological Data, the other 4 having dealt with blood, nutrition and metabolism, respiration, and miscellaneous "biological data," respectively. The book covers the anatomy, chemical composition and physical properties, normal physiology (blood volumes, cardiac output, heart rate, blood pressure, blood flow), electrocardiography, effects of stress (pregnancy, varying O_2 and CO_2 concentrations, compression, and acceleration, radiation), pharmacology, and pathology.

EMOTION AND PERSONALITY. By Magda B. Arnold. Volume 1, Psychological Aspects. xiv + 296 pp.; Volume 2, Neurological and Physiological Aspects. xi + 430 pp. Columbia University Press, New York, 1960. $7.50 each volume. In the author's words, this work "represents an attempt to develop a theory of emotion and feeling that can adequately integrate the psychological, neurological, and physiological aspects of affective phenomena and place them in their proper perspective as important factors in personality integration." While a large amount of research on the physiological components of emotion has been conscientiously gathered and sifted, the author relies basically on the phenomenological (experiential) analysis of emotions as the guide for developing a comprehensive theory of feelings and emotions.

THE INTELLIGENT MAN'S GUIDE TO SCIENCE. By Isaac Asimov. Volume I. The Physical Sciences. xiv + 302 pp. Volume 2. The Biological Sciences. viii + 853 pp. Basic Books, New York, 1960. $15.00. As George W. Beadle tells us in his introduction, the book is designed for "persons who want to know what science is all about, how it came to be, and who are some of the people responsible for its development." The body, the species, the mind are all there, vividly presented—a popular account yet reliable and up-to-date.

PRINCIPLES OF GENERAL PHYSIOLOGY: VOLUME 2. GENERAL PHYSIOLOGY. By L. E. Bayliss. xxiii + 848 pp. Longmans, London and New York, 1960. $17.50. The first edition, from the pen of W. M. Bayliss, saw the light of the day in far-off 1915. The fifth edition grew into 2 volumes and was written by the son of the author, with skill and care. The chapters on growth, heredity, and adaptation and evolution are "general" enough to be of interest to scientists outside the narrower physiological fraternity.

DICTIONARY OF WORD ROOTS AND COMBINING FORMS. By Donald J. Borror. v + 135 pp. N-P Publications, California, 1960. $2.00. The sub-title informs us that the dictionary was "compiled from the Greek, Latin, and other languages, with special reference to biological terms and scientific names." The taxonomist will find useful the sections on the formation of scientific names and on some common characteristics (color, size, shape, etc.) for which Latin and Greek forms are given, but the life of all biologists would have been much simpler if they had had the chance to memorize in high school these 12-odd pages and the opportunity to consult later a dictionary such as the present one.

STRUCTURE AND FUNCTION OF MUSCLE. Edited by G. H. Bourne. Volume 2, Biochemistry and Physiology. xiv + 593 pp. $16.50. Volume 3, Pharmacology and Disease. xiv + 489 pp. $15.00. Academic Press, New York, 1960. The trilogy is an outstanding reference work. The second volume includes, among many other topics, the physiology of muscular exercise and human fatigue; the third volume, the effects of nutritional deficiencies upon muscle and age changes in muscle.

THE CENTRAL NERVOUS SYSTEM. Edited by Mary A. B. Brazier, 475 pp. The Josiah Macy, Jr. Foundation, New York, 1960. $7.50. Transactions of the third of the outstanding Macy conferences on this topic. Of special relevance is a large chapter (pp. 33-126) by H. W. Magoun, Louise Darling, and J. Prost on the evolution of man's brain.

FUNCTIONAL NEURO-ANATOMY. By A. R. Buchanan. 377 pp. Lea and Febiger, Philadelphia, 1961. $8.50. The fourth edition of a basic textbook. A special feature of the book is the extensive atlas of brain stem and adjacent structures (pp. 269-343).

PIGMEOS EN AMÉRICA? (Are there pygmies in America)? By Juan Comas. 54 pp. Cuadernos del Instituto de Historia, Serie Anthropológica No. 9. Universidad Nacional Autónoma de México, 1960. No price indicated. The author surveys the evidence, provided by historical and contemporary literary sources, and concludes that 1. there is a large number of aboriginal tribes in the Northeast of South America with short stature (150 to 159 cm for males, 140 to 148 cm for females); 2. in three tribes—Yupa, Ayamanes, Shishirana—true dwarfs (adult stature below 150 cm in males, below 140 cm in females) are relatively frequent. Adequate biological investigations regarding the "how" and "why" of this phenomenon are lacking.

STYLE MANUAL FOR BIOLOGICAL JOURNALS. Prepared by Committee on Form and Style of the Conference of Biological Editors. viii + 92 pp. American Institute of Biological Sciences, Washington, 1960. $3.00. In science a great deal of emphasis is being placed on standardization. At the same time, there has been an utter chaos as regards the technical aspects of scientific writing, such as the character and the placement of the summary, use of abbreviations, or citation of references. The present volume is the

outcome of an endeavor to achieve greater uniformity in manuscripts prepared for publication in biological journals. Happily, *Human Biology* is among the journals which adopted the principles put forth in this manual. In the next edition instructions regarding the transliteration of non-Latin alphabets (esp. of the Cyrillic alphabet used in Russian) would be a useful addition.

THE DEVELOPMENT OF BONY PELVIC SEXUAL DIMORPHISM IN MICE. By E. S. Crelin. Ann. N. Y. Acad. Sci., Volume 84, Art. 13, 33 pp. 1960. No price indicated. Experimental analysis of factors controlling body build is one of the avenues of progress in animal (and human) morphology, a proverbial "shot in the arm" needed—according to a concerned critic—"if physical anthropology is not to continue, as it seems to be doing, along a moribund course." Two approaches were used to demonstrate that androgen is the factor responsible for the production of bony pelvic sexual dimorphism: 1. Mature male and female mice, gonadectomized at birth, have pelves of the female (in reality: agonadal) type. 2. When mice gonadectomized at birth are treated with testosterone during development, both males and females have male-type pelves when adult.

HUMAN NUTRITION, HISTORIC AND SCIENTIFIC. Edited by I. Galdston. xvii + 321 pp. International Universities Press, New York, 1960. $6.00. A broad coverage of topics in the science of nutrition and its applications, spanning the biological and social sciences: "Nutrition is still a young, growing, and vigorous science with increased value to mankind as a result of each discovery. Narrow definitions of nutrition are no longer acceptable" (C. G. King). Food habits in different parts of the world, the historical development of the science of nutrition, nutrition and population pressures, epidemiology of protein malnutrition—these are some of the dishes on the richly laden table.

SPACE BIOLOGY. By James Stephen Hanrahan and David Bushnell. vi + 263 pp. Basic Books, New York, 1960. $6.00. Since no living organisms have yet been retrieved from the extraterrestrial realm, "space biology"—as the subtitle indicates—deals with "the human factors in space flight." Specifically, the volume is concerned with the space vehicle (cabin atmosphere, alimentation and waste), g-forces and weightlessness, and the radiation hazards. The authors attempted, successfully, to present two aspects of the story of the progress toward manned space flight: 1. the highlights of our present understanding of the complex problems of space flight, and 2. a historical survey of the developments leading to the current stage in man's preparation for going into space.

DIMENSIONS OF MIND. Edited by Sidney Hook. xiii + 281 pp. New York University Press, Washington Square, 1960. $5.00. The title suggests a mathematical analysis (vector, factor analysis) of "mental abilities" but this topic is not presented at all. The proceedings of the 3rd Annual New York University Institute of Philosophy are centered around 3 subjects: 1. The mind-body problem, 2. The brain and the machine, and 3. Concept formation.

COLD INJURY. Edited by Steven M. Horvath. 375 pp. The Josiah Macy, Jr. Foundation, New York, 1958. $6.50. In recent years a great deal of work was done on man's responses to cold, a problem that earlier was studied much less intensively than the effects of high environmental temperatures. The volume gathers together, in a welcome fashion, the information obtained in field studies (Operation Deep Freeze and other experiences in the Antarctic and in Australia), human laboratory investigations (energy balance and heat production, including shivering and exercise), and animal studies (removal of the hypothalamic "thermostat" in dogs, deep hibernation in rodents).

THE BIOLOGIST'S HANDBOOK OF PRONUNCIATIONS. By Edmund C. Jaeger, xvi + 317 pp. C. C. Thomas, Springfield, Illinois, 1960. $6.75. Dr. Jaeger is well known as the author of "A Source-Book of Biological Names and Terms" and "A Source-Book of Medical Terms," both published by Thomas. While the meaning of some entries in the present volume is indicated, the principal aim is to aid biologists with the accentuation and other aspects of the pronunciation of Greco-Latin technical terms.

PSYCHOLOGY: A STUDY OF A SCIENCE. Edited by Sigmund Koch. Study 1. Conceptual and Systematic. Volume 1. Sensory, Perceptual and Physiological Formulations. x + 710 pp. Volume 2. General Systematic Formulations, Learning, and Special Processes. vii + 706 pp. Volume 3. Formulations of the Person and the Social Context. x + 837 pp. McGraw-Hill Book Company, 1959. No prices indicated. Actually, it will be Volume 4, entitled "Biologically Oriented Fields: Their Place in Psychology and in Biological Science" which will be of special interest to biologists concerned with behavior (and to psychologists interested in behavior's biological "substructure"). Nevertheless, we can not bypass in silence the first 3 volumes. For one, biological (physiological) considerations are not lacking (*e.g.*, C. T. Morgan's "Physiological theory of drive," in Volume 1; R. A. Hinde's chapter on the field study of animal behavior, in Volume 2; F. J. Kallmann's "Psychogenetic studies of twins," in Volume 3). More importantly, perhaps, this series of 7 volumes will be a unique portrait of contemporary American psychology, a welcome basis for a critical examination of the current status of scientific study of behavior.

FITTING THE JOB TO THE WORKER. Reported by Bernard Metz. 115 pp. European Productivity Agency, Paris, 1960. $1.25. This is a general report about an international conference held in Zurich, Switzerland, on 2nd to 7th March 1959, under the sponsorship of the Organization for European Economic Cooperation. Small in size, the volume is an effective mirror of contemporary science of human work, interdisciplinary in nature. The anthropometric and biomechanical requirements of work-place design were discussed by H. Schnewlin (Switzerland, p. 29) and J. Weiner (Oxford, England, p. 32).

AN INTRODUCTION TO EXPERIMENTAL DESIGN. By William S. Ray. x + 254 pp. Macmillan, New York, 1960. $6.50. The volume emerged, in time, from lecture notes for a one-semester course, given to graduate students in psychology at Pennsylvania State University and concerned with "issues, principles and techniques peculiar to those investigations in which the control of natural processes is actually attempted and directly observed."

ERROR AND DECEPTION IN SCIENCE. By Jean Rostand. 195 pp. Basic Books, New York, 1960. $4.00. The book borrows the title from the first chapter but the theme is not pursued in the five "essays on biological aspects of life," roaming freely from repercussions of the biological findings in law, through considerations of effeminate men and masculine women, to the value of the motion-picture technique in biology. Well-informed yet not pedantic, this is "light reading," for the most part. However, the power of preconceived ideas, basic to large-scale delusions and what Rostand calls "collective errors" in science, documented in the first chapter, can not be taken lightly. Myth-making is a powerful tool in the service of self-seeking charlatanism and ideological dogmatism.

MEDICAL PHYSIOLOGY AND BIOPHYSICS. Edited by Theodore C. Ruch and John F. Fulton. xxii + 1232 pp. Saunders, Philadelphia, 1960. No price indicated. "An American Text-book of Physiology," edited by William H. Howell and published in 1896 was the great-great-grandfather of this (18th) edition. Subsequently, the title was changed three times, with the 16th and 17th editions coming back to the initial formula except for the deletion of the geographic reference. The newest metamorphosis reflects two opposing tendencies: On the one hand, increased concern with pathology (clinical applications), on the other hand, stronger emphasis on "university disciplines," especially physics. It appears most peculiar that the index lists "unconditioned" but not "conditioned" reflexes. Was not I. P. Pavlov's paper on "The scientific investigations of the psychical faculties or processes in higher animals" published in English, in *Science* (24: 613-619) in 1906?

DEVELOPMENT OF THE PERCEPTUAL WORLD. By Charles M. Solley and Gardner Murphy. xi + 353 pp. Basic Books, New York, 1960. $6.50. An attempt to weave a new cloth from the fibers constituting the psychology of the learning process and the process of perceiving: "We may learn to understand perception better if we see its parameters defined in terms of learning as well as in terms of structural components and physiological units which come with the living package itself. We may understand learning better if we realize the role that a plastic and modifiable process of perceiving may impose upon basic motor learning." The authors note that the biological significance of perceptual learning lies in the flexibility, adaptability it gives to the perceiving organism, thus increasing the likelihood of survival (p. 33). Chapter 12, entitled "The signals within us: Internal scanning and feedback," is devoted to the role of proprioception ("the language of our muscles") and enteroception.

DISCUSSIONS ON CHILD DEVELOPMENT. Edited by J. M. Tanner and Bärbel Inhelder. xiii + 186 pp. Tavistock Publications, London, 1960. 30s net. These are The Proceedings of the Fourth (and last) Meeting of the World Health Organization Study Group on the Psychobiological Development of the Child. The meeting was held in Geneva in 1956. The volume is centered around Jean Piaget's paper on "The general problems of the psychobiological development of the child" (main factors affecting development—heredity, physical environment, including nutrition, social environment and, as the fourth factor, equilibration; stages of development; and mechanisms of development).

MEDICINE AND BIOLOGY IN EXPERIMENTS ON MONKEYS. Edited by I. A. Utkin. Trans. from Russian by Ruth Schachter. vii + 276 pp. Pergamon Press, New York, 1960. $7.50. The Institute for Experimental Pathology and Therapeutics, affiliated with the Academy of Medical Sciences of the U. S. S. R. and located at Sukhumi, in Georgia, is well known for its facilities for medico-biological research on monkeys. In the present volume emphasis is placed on the study of conditioned responses ("higher nervous activity") and infectious pathology. Primatologists and human biologists may wish to acquaint themselves with the work on seasonal (and diurnal) fluctuations in the red blood cells.

THE DELAYED EFFECTS OF WHOLE-BODY RADIATION. Edited by Bernard B. Watson. ix + 80 pp. The Johns Hopkins Press, for the Operations Research Office, Baltimore, Maryland, 1960. $4.50. While a considerable amount of information has been obtained on short-term hazards of nuclear radiation, less well known are the delayed effects of chronic exposures to low levels of radiation which fail to produce clinical symptoms of radiation sickness. Among these effects, two categories are of special interest to readers of *Human Biology*: the life shortening (i. e., accelerated rate of aging) and genetic alterations. In the present symposium two papers deal with the life-span effects, and one paper is concerned with genetic effects in animals and man.

BALANCING THE HUMAN HEMOGLOBIN POLYMORPHISMS

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IN recent years it has become well accepted that the high frequencies of the sickle cell gene are maintained by a selective advantage on the part of the heterozygotes for this gene and that this selective advantage is a greater resistance to falciparum malaria. In Africa, India, and the Middle East, where human populations with high frequencies of the sickle cell gene have been found, there is evidence for these populations having been exposed to intense malaria due to *Plasmodium falciparum*, the most deadly of the human malaria parasites. And in addition the little experimental work that has been done tends to confirm this relationship between falciparum malaria and the sickle cell trait carriers (heterozygotes). Thus, in the face of the adverse selection due to the early death of homozygotes for the sickle cell gene, high frequencies of this gene are maintained in these tropical regions by the selective advantage of the sickle cell heterozygotes in a malarious environment; and hence the sickle cell locus appears to be the first clear evidence for balanced polymorphism in human populations (for reviews of the sickle cell problem, see Allison, 1957; Neel, 1956; Vandepitte, and Delaisse, 1957).

Although the sickle cell gene is the only hemoglobin gene for which there is good evidence for balanced polymorphism, there are several other genes which produce similar hemoglobin abnormalities and which are found like the sickle cell gene in appreciable frequencies in many populations who inhabit the tropical regions of the Old World. Thus, there has been a tendency to assume that high frequencies of other abnormal hemoglobin genes are also maintained by an increased resistance of the heterozygotes to malaria. These genes are: thalassemia, which is distributed from Portugal to the Philippines although these occurrences may consist of several "genes" (Zuelzer, Neel, and Robinson, 1956; Ingram and Stretton, 1959); hemoglobin C, which is restricted to West Africa and parts of North Africa; and hemoglobin E, which is found from about Calcutta through Southeast Asia and Indonesia to the Moluccas. When homozygous or in combination with one another, these genes produce

clinical conditions which considerably lessen the viability of the individual. Thus, for the genes to attain appreciable frequencies, there must be either a high net mutation rate to them from the normal allele or positive selection for the genes. Since there is no evidence for high mutation rates, positive selection seems to be the most likly answer; but with the exception of Ceppellini's (1955) work on thalassemia in Sardinia there is little evidence for this postitive selection. However, both analogy with the sickle cell gene and Ceppellini's work do seem to indicate malaria as the balancing factor. In this paper I would like to explore further the analogy with the sickle cell gene and then propose several hypotheses as to how the high frequencies of these abnormal hemoglobin genes are maintained. Although much of the following discussion is admittedly speculative, it seems to me that pursuing the logical implications of the sickle cell polymorphism can provide considerable insight into the nature of the other hemoglobin polymorphisms and particularly those of thalassemia, hemoglobin C, and hemoglobin E.

There are two aspects of the sickle cell polymorphism on which there is still disagreement and which have considerable bearing on the analogy between the sickle cell and the thalassemia, hemoglobin C and hemoglobin E genes. These two aspects are quite interrelated and not independent and can be briefly characterized as (1) the manner by which the heterozygotes for the sickle cell gene have a selective advantage, and (2) the nature of the physiological interaction between the sickle cell gene and the falciparum malaria parasite which confers this selective advantage. With regard to the first question or aspect there are two possible methods by which the heterozygotes may have a selective advantage, either by a greater fertility or a greater survival to adulthood. Allison (1957) considers the answer to lie in a relative immunity to falciparum malaria and hence it is a greater survival of the heterozygotes, while I (Livingstone, 1955) have advanced the possibility of the female heterozygotes for the sickle cell gene having a greater fertility. The evidence for the sickle cell heterozygotes having a relative resistance to falciparum malaria or at least not dying as frequently from the disease is quite convincing for certain localities, Uganda (Raper, 1956), and the Belgian Congo (Lambotte-Legrand and Lambotte-Legrand, 1958; Vandepitte and Delaisse, 1957), while evidence for such an immunity has not been forthcoming in other localities such as Northern Ghana (Colbourne and Edington, 1956) or Nigeria (Walters and Bruce-Chwatt, 1956). It does seem to be a fact that the areas where the evidence has been found seem to have less malaria than those where such an immunity has not been

demonstrated, and in addition, among the Baamba, who have the highest recorded frequency of the sickle cell gene, falciparum malaria does not seem to be holoendemic according to the parasite rates recorded by Lehmann and Raper (1956). The evidence for the sickle cell heterozygotes having a greater fertility is much less (Allard, 1955; Delbrouck, 1958; and Livingstone, 1955), but not as many investigations have been carried out on this aspect of the problem. In any case the maintenance of the high frequencies of the sickle cell gene is most likely due to a combination of these two interactions and perhaps to other factors. The question then arises as to the physiological mechanism by which the heterozygotes acquire their selective advantage.

Allison (1957) and many others have maintained that the falciparum malaria parasite is unable to metabolize sickle cell hemoglobin as easily as normal hemoglobin and thus the parasite will not develop as well in persons with sickle cell hemoglobin. This inability of the parasite to metabolize sickle cell hemoglobin is attributed to the lack of appropriate proteolytic enzymes. Heterozygotes for the sickle cell gene usually have about 40% sickle cell hemoglobin and the rest, normal hemoglobin, while homozygotes for the gene or those simultaneously heterozygous for the sickle cell and some thalassemia genes will have most of their hemoglobin of the sickle cell variety. Raper's (1959) recent experiments to test this explanation have failed to produce any convincing evidence for it. Although homozygotes for the sickle cell gene, who have the most sickle cell hemoglobin, also had the lowest mean parasite density, and the heterozygotes for the gene, who are intermediate in the composition of their hemoglobin, were also intermediate in mean parasite density, other explanations in addition to the "inability of the falciparum parasite to metabolize sickle cell hemoglobin" theory can be found for this phenomenon. Thus, the crucial evidence from these experiments seems to be that although Raper found that the falciparum parasite could only be cultured for one cycle, it could be grown on sickle cell hemoglobin as easily as on normal hemoglobin and thus the parasite appeared to metabolize both hemoglobins equally well.

In addition to the absence of positive evidencs for the "inability to metabolize" theory, it seems to me that when our knowledge of the abnormal hemoglobins as a whole is considered, there is much circumstantial evidence against the theory. By circumstantial evidence I mean that the implications of this theory both for the distribution of the other abnormal hemoglobins and for the reaction of the other human malaria parasites to sickle cell hemoglobin do not seem to accord with

the facts. Of course this evidence against the theory is only in terms of what one would expect assuming the theory to be true, but I would like to emphasize that in no case do these expectations seem to be fulfilled.

If it were simply the inability of the parasite to metabolize sickle cell hemoglobin, one would first of all expect the parasites of sickle cell trait carriers to exhibit abnormal morphology as an indication of their poor or "starved" condition, but these parasites seem to be quite natural in appearance. Even the gametocytes, which are the only mature parasites found in the peripheral blood, are normal in appearance and have as high a density in sickle cell trait carriers (Edington and Laing, 1957). Secondly, if the falciparum parasite could not metabolize sickle cell hemoglobin, one would expect the other malaria parasites to have the same difficulty; but there is no evidence that the sickle cell gene protects its carriers against the other species of human malaria parasite (*P. malariae*, *P. vivax*, and *P. ovale* are the usually accepted ones). In fact on some occasions (Allison, 1954; Colbourne and Edington, 1956) higher infections of *P. malariae* have been found in sickle cell trait carriers. Thirdly, if the falciparum parasite could not metabolize sickle cell hemoglobin, it should have difficulty metabolizing other abnormal hemoglobin. But Edington and Laing (1957) failed to find any evidence that hemoglobin C protects its carriers against falciparum malaria. Of course it could be argued that hemoglobin S is the only one with the particular configuration which the falciparum parasite is unable to metabolize, but this seems to be quite unlikely particularly since hemoglobin C is a replacement of the same amino acid as hemoglobin S and both only differ from normal hemoglobin by this one amino acid substitution. Finally, if it is the abnormality of the hemoglobin *per se* which confers an immunity to falciparum malaria, one would expect that the many abnormal hemoglobin genes with minor morphological and physical abnormalities would tend to replace the sickle cell gene with its very deleterious side effects. Since the deleterious effects of the genes responsible for most other abnormal hemoglobins appear to be much less than those of the sickle cell gene, these genes would tend to build up to very appreciable frequencies with very little positive selection for them. However, there are many reports of very low frequencies of hemoglobins D, G, I, J, K, L, N, O, and J in populations with holoendemic falciparum malaria, and there seems to be no tendency for these hemoglobin genes to replace the sickle cell gene. It should also be noted that *none* of the hemoglobin genes with minor morphological abnormalities associated with them

attain frequencies anywhere which approach those of the sickle cell, thalassemia, hemoglobin E, or hemoglobin C genes. Thus, it can be seen that the genes with the most deleterious side effects are those with the highest frequencies. Such a situation seems to me to be completely unexplainable in terms of the "inability to metabolize" theory. But although this state of affairs is paradoxical in terms of the latter theory, if the immunity to malaria which the hemoglobin genes appear to confer had a physical or morphological basis, this would be expected. According to such a theory, those genes which caused the red cells of its carriers to be more different from normal would confer more of an immunity, although of course in any particular situation it would depend on whether the particular cytological abnormality interfered in any way with the parasite in question.

Such an explanation for the sickle cell polymorphism was first advanced by Miller, Neel, and Livingstone (1956) and is based on the fact that this particular abnormal hemoglobin results in certain physical changes in the red blood cell which interact with the falciparum parasite in such a way as to inhibit its development. Miller, Neel, and Livingstone (1956) reasoned as follows: *Plasmodium falciparum* is the only human malaria parasite in which the mature asexual forms are not seen in the peripheral blood. As the parasite matures, the membrane of the red cell which contains the parasite develops a "stickiness" and the parasitized cells adhere to each other and to the walls of the blood vessels. This occurs mostly in the inner organs. Since this is a relatively anoxic environment the red cells of sickle cell trait carriers would tend to sickle, and thus interfere with the development of the parasite. Thus, this interference with the life cycle of P. falciparum in the the inner organs would be the basis of the relative immunity of the sickle cell trait carriers. Since cerebral malaria, which is the usual cause of death from falciparum malaria, is the result of the clogging of the capillaries of the brain by parasitized red cells, the interruption of the falciparum life cycle in the brain of sickle cell trait carriers would be the reason for fewer deaths among them from *P. falciparum*. Since heavy falciparum infections of the placenta are the cause of much prematurity, stillbirth, and neonatal death, the interruption of the development of *P. falciparum* in this organ would result in a greater fertility on the part of the sickling females. Thus, this theory provides an explanation of how falciparum malaria could result in the sicklers having both a higher survival and a higher reproduction rate. Since *P. falciparum* is the only one of the human malaria parasites which develops in the anoxic environment of the inner

organs, this theory can also provide an explanation of why the sickle cell trait carriers do not have a relative resistance to the other malaria parasites.

This theory also has implications for the other abnormal hemoglobins, but it is not a simple analogy. Sickle cell hemoglobin is the only one which behaves in such a way in an anoxic environment so that the interaction between it and the falciparum parasite is unique. However, if it is the physical peculiarities of the red blood cells of sickle cell trait carriers which give them their advantage, then it may well be the same for the other abnormal hemoglobins. Thus, if we list the cytological and physiological abnormalities associated with each hemoglobin, perhaps some explanation of its selective advantage will be forthcoming. This approach may also prove productive for another previously mentioned reason. Since the abnormal hemoglobin genes which have the least physical abnormality are those which are found in very low frequencies, there seems to be less selection for these genes. Thus, the amount of selection for the hemoglobin genes is correlated with the number or magnitude of the physical abnormalities of the red blood cell.

For the remainder of this paper the analogy with the sickle cell gene will be pursued only for the thalassemia, hemoglobin C and hemoglobin E genes, the three other abnormal hemoglobin genes which attain appreciable frequencies. The only abnormality which heterozygotes for any one of these genes and the normal allele seem to have in common is an increased resistance of their red cells in hypotonic salt solution. This osmotic resistance is more pronounced and a more constant finding in thalassemia but it does seem to be present in most heterozygotes for either hemoglobin C or E. In many cases the values for the latter two heterozygotes are within the limits of normals but almost always are near the lower limit. An increased osmotic resistance of the red cells is usually thought to be the result of an altered surface/volume ratio of the cells. For both the thalassemia and hemoglobin E heterozygote this altered surface/volume ratio seems in turn to be a result of a microcytemia which is a feature of these conditions but not of heterozygosity for the hemoglobin C gene. Like the increased osmotic resistance, the microcytemia in thalassemia is more pronounced than that associated with hemoglobin E, which is rather minimal and usually within normal limits. For example Luan Eng (1956) found the mean cell volume for normal healthy adult male Indonesians to be 86.9 and that for healthy adult male carriers of hemoglobin E to be 78.8, a difference which is statistically significant ($t = 5.27$, 41 d.f., $P << .001$). For hemoglobin C

the increased osmotic resistance of the red cells also seems to be the result of an altered surface/volume ratio but it is not a microcytemia. Instead heterozygosity for this gene is usually characterized by an increased number of target cells, which, although of normal volume, appear to be flatter with a more uneven surface and hence have a greater surface area. However, for hemoglobin C and perhaps for the other two conditions as well, there is another possible factor which contributes to the increased osmotic resistance of their red cells. Younger red cells are known to be more resistant than older cells in hypotonic salt solution (Marks, Johnson, Hirschberg, and Banks, 1958). Since they are also larger, this indicates that factors other than surface/volume ratio are involved in determining osmotic resistance. In any case, however, there is some evidence for heterozygotes for hemoglobin C having a decreased red cell survival time (Kaplan, Zuelzer, and Neel, 1953), so that the red cell populations of these individuals would contain more young red cells and hence would have a greater osmotic resistance. But it should be noted that the evidence for heterozygotes for hemoglobin C having a decreased survival time is quite equivocal and more work needs to be done on this aspect of the problem. In summary, heterozygosity for either of these three genes results in an altered surface/volume ratio, which for thalassemia and hemoglobin E is due to a microcytemia, and for hemoglobin C is due to an increase in target cells. In addition there is a possibility of a decreased survival time for hemoglobin C and perhaps for the others as well. The problem then becomes how these conditions or abnormalities could influence an individual's reaction to malaria.

Immunity to malaria is due primarily to the ability of the phagocytes to engulf the malaria parasites, and Taliaferro (1949) has shown that this occurs to a great extent when the parasite is within the red blood cell. Taliaferro (1949) has divided the course of the development of an immunity into two stages. The first he calls the stage of innate immunity for which I will use the term, innate resistance, and the second, the stage of acquired immunity. The first response or the innate resistance of an organism to malaria is a tremendous increase in the amount of phagocytosis of red blood cells by both the macrophages of the spleen, liver, and bone marrow, and the microphages of the peripheral blood. During this stage of innate resistance phagocytosis is somewhat non-specific and both parasitized and non-parasitized red cells are engulfed, but later in the stage of acquired immunity phagocytosis becomes much more specific for red cells which contain parasites. However, during the stage of acquired immunity the parasite within the red cell has to reach a certain

maturity before the parasitized cell can be indentified by the phagocyte; hence the increase in specificity of phagocytosis is concerned only with parasites in the later stages of maturity. Concomitant with the development of specificity of phagocytosis, there is also evidence for the development of humeral factors which assist this phagocytosis (Zuckermann, 1945). It can thus be seen that the ability of the phagocyte to identify parasites within the red cells is the most important aspect of the development of an immunity to malaria. During the stage of acquired immunity an opsonin seems to be involved and the phagocytosis is mostly by the macrophages of the spleen, liver, and bone marrow. But since the stage of innate resistance to malaria occurs before the development of humeral factors, the identification of parasitized red cells by both these macrophages and the microphages of the peripheral blood appears to be a rather mechanical or physical process. During this stage the host is able to eliminate about 66% of the parasites but the rapid multiplication of the remaining 33% still is able to bring on an acute crisis. Whether or not an individual is able to survive a malaria attack depends on whether the acquired immunity develops in time to prevent lethal concentrations of the parasite, and this in turn depends on whether an individual's innate resistance can cut down the multiplication of the parasite sufficiently to give the acquired immunity time to develop. There do appear to be individual differences in the amount of innate resistance, and the sickle cell gene undoubtedly contributes to these differences. Thus, the problem becomes how the abnormalities associated with the three abnormal hemoglobin genes under discussion could influence the innate resistance of their carriers. It should be noted that the effect would have to be rather small. If, for example, these individuals could eliminate 70% of the parasites at each growth cycle instead of the normal 66%, the effect would be tremendous and delay the attainment of lethal concentrations of the parasite by several days. It should be noted here that the figure of 66% is quoted from Taliaferro (1949) and is for the development of a malarial infection in a howler monkey not a human, but the progress of malaria is much the same in man although the percentages probably differ.

As was previously stated, phagocytosis during the stage of innate resistance appears to be a physical process. Now in individuals whose red blood cells had a greater surface area per unit volume, the probability of an intracellular parasite being close to, touching, or even distorting the red cell membrane or wall would be greater than in individuals with a normal surface/volume ratio. Thus, it seems to me

that the phagocyte would have a greater probability of identifying or recognizing parasites in these individuals' red blood cells. In addition if the red cells were smaller and flatter as are those in microcytemia the parasite would have even a greater probability of being recognized. Hence these two conditions could very easily enhance phagocytosis during this stage of innate resistance when there are no humeral factors present. In this manner individuals who are heterozygous for either thalassemia, hemoglobin E or hemoglobin C may well have an increased innate resistance to malaria; and since this is the basic immunity process to all human malarias, they would have an increased innate resistance to all species of human malaria parasites.

There are other possible ways in which these conditions may contribute to an increased resistance to individual species of human Plasmodia. Plasmodium vivax selectively attacks reticulocytes and larger red blood cells and also enlarges the red cell as it matures (Maegraith, 1948). Individuals heterozygous for either thalassemia or hemoglobin E, because of their microcytemia, may be refractory to infections with P. vivax; perhaps due to the inability of their smaller cells to contain the mature parasite or to the inability of the mature parasite to obtain sufficient nutrition from these smaller cells. While P. vivax attacks reticulocytes and P. falciparum is able to attack red cells of all ages, another human Plasmodia, P. malariae, selectively attacks older red blood cells. Since individuals heterozygous for hemoglobin C appear to have a shortened red cell survival time, they would have fewer old red cells and hence may be more resistant to infection with P. malariae.

If the foregoing hypotheses have any validity, then there are implications for the world distributions of these abnormal hemoglobins; but the general implication is not that there is a simple and direct association between the distribution of malaria and the distributions of these abnormal hemoglobins. The presence of the selective advantage of a particular gene in an area does not imply that the gene should be present in high frequency. Other factors, such as how long the advantage has been present, how much gene flow there has been, and whether or not favorable mutations have occurred must also be considered. Thus, the logic of the situation is that wherever high frequencies of these abnormal hemoglobins are found, there should also be evidence of malaria being a strong selective factor, but not that wherever there is malaria, there should be high frequencies of abnormal hemoglobins. No attempt will be made here to discuss in detail whether the world distributions of these hemoglobins accord with these possible selective advantages, but there

do not appear to be any striking contradictions. And in addition it seems to me that these hypothesized selective advantages do explain some of the peculiarities of the distributions of these abnormal hemoglobins.

Thalassemia is the most widespread hemoglobinopathy and with the exception of Australasia is found in virtually all the malarious regions of the Old World. This spread would be expected since thalassemia appears to consist of several different "genes" and hence one would expect to encounter more mutations for this condition. According to the hypotheses of this paper thalassemia also has the most widespread selective advantage since it appears to enhance the resistance of its carriers to all species of human malaria; so that one would expect thalassemia to be very widespread for this reason. In Macedonia and Sicily where much of the malaria was due to P. falciparum, both the sickle cell and thalassemia genes are found. But in the Po River Valley of Northern Italy where P. vivax is the predominant parasite, only thalassemia is found in high frequencies. On the other hand the distributions of hemoglobins C and E are much more restricted, and both appear to be due to the spread of a single mutation. Like thalassemia, hemoglobin E is widespread in Southeast Asia, but thalassemia seems to be present in appreciable frequencies in China and the Philippines while hemoglobin E is not. To the south, both have similar distributions in Indonesia and both are also absent from New Guinea and other parts of Australasia where there is at present holoendemic malaria. However, there is some evidence (Lambert, 1949) for the recent spread of malaria in these regions.

The distribution of hemoglobin C is even more restricted than that of hemoglobin E, and is further complicated by the fact that all populations with appreciable frequencies of hemoglobin C with the possible exception of the Berbers (Pasternak and Roberts, 1956) also have appreciable frequencies of the sickle cell gene. The hemoglobin C gene is an allele of the sickle cell gene and the two are thus in competition. Since persons who are heterozygous for the sickle cell and hemoglobin C genes have a rather severe anemia and hence a lessened viability, these two genes would tend to drive each other out of a population even in the presence of a selective advantage for both. An increase in either S or C gene would very likely involve a decrease in the other. In fact if the only change which caused an increase in one of these genes is an increase in the selective advantage of the heterozygote for this gene and the normal allele, then mathematically we can prove that this must result in a decrease in the other gene. Allison (1956) has also emphasized that

these two genes would tend to be mutually exclusive and has shown that in the population of West Africa the sum of the frequencies of the S and C genes tends to be a constant, although there appear to be some exceptions to this rule (Rucknagel and Neel, 1960). Thus, wherever the sickle cell gene has a greater selective advantage it would be able to replace more of the hemoglobin C gene.

Now as was noted previously, the sickle cell gene confers a selective advantage to but one of the human malarias, that due to *P. falciparum*. This is the most severe of the malarial diseases and undoubtedly is the cause of most deaths from malaria. On the other hand the abnormalities associated with heterozygosity for hemoglobin C appear to confer an increased innate resistance to all species of human malaria and perhaps increase the resistance to *P. malariae* to a greater extent than to the others. Hence one would expect high frequencies of hemoglobin C in areas where *P. malariae* is particularly prevalent and high frequencies of the sickle cell gene in areas where *P. falciparum* is the predominant parasite. The highest frequencies of hemoglobin C are found in the region of the bend of the Niger River, precisely where *P. malariae* is "particularly prevalent" (Wilson, 1949), but even throughout West Africa there is not a direct association between high frequencies of *P. malariae* and of the hemoglobin C gene. This is due to the fact that all human malarias are transmitted by anopheline mosquitoes, so that where transmission is greatest, all species of malaria will tend to attain their highest frequencies. However, there are some factors which contribute to differences in transmission. *P. malariae* is a low-density, long-lasting infection and will thus be transmitted to a greater extent than *P. falciparum* when transmission is decreased, although of course with a decrease in transmission both species will decrease in absolute numbers. In the tropical forest regions of Africa, where transmission of malaria is greatest and occurs throughout the year, *P. falciparum* is the predominant parasite but *P. malariae* is also present in a considerable frequency. As one proceeds north out into the sudan and then into the desert, however, *P. malariae* becomes increasingly important and it is the predominant parasite in the oases of the Sahara (Hackett, 1949). Amongst those populations which have hemoglobin C, the frequencies of both the S and C genes seem to correlate with this cline in malarial predominance. In the tropical forest regions of Southern Nigeria and Ghana, *P. falciparum* is the major parasite and high frequencies of the sickle cell gene are found; *P. malariae* is also quite common and hence lower frequencies of hemoglobin C are found. In Northern Ghana, the

frequency of *P. malariae* is much higher (Colbourne and Edington, 1956) but *P. falciparum* remains the predominant parasite. Under these conditions, however, hemoglobin C attains its highest frequency while the sickle cell gene decreases to about 5% from its high of 10% in Southern Ghana. On the other hand in Nigeria the frequency of *P. malariae* does not seem to increase in much of the Northern region and in general has a much lower frequency throughout so that the sickle cell gene remains at its high frequency and there is little hemoglobin C present (Edington, 1959). *P. malariae* attains its highest frequency in the Cameroons in the mountains of the north near Lake Chad (Languillon, 1958) and the Wakura who inhabit these mountains are the only tribe in that area to have any hemoglobin C and they have about 6% heterozygotes for the gene (Roberts and Lehmann, 1958).

Although in the previously discussed areas of West Africa there appears to be some association between hemoglobin C and S and the *Plasmodium falciparum* and *malariae*, for the rest of West Africa this does not appear to be so. However, in most of these other areas of West Africa, hemoglobin C is found in very low frequencies. In Eastern Nigeria among the Ibo and in the Southern Cameroons there is almost no hemoglobin C although *P. malariae* is present. Along the West Coast of West Africa from Liberia to the Senegal there is relatively little hemoglobin C but in many places *P. malariae* is quite prevalent. The highest frequency of hemoglobin C (5%) has been recorded from Sierra Leone (Allison, 1956), which is partly tropical rain forest and partly savannah, and *P. malariae* has been recorded from there in very high frequencies among the more primitive populations (Conran and Conran, 1956). However, *P. malariae* is also quite common in the Gambia and Senegal, but the frequencies of hemoglobin C are low (1%).

Hemoglobin C seems to have spread across the Sahara to Morocco and Algeria to a greater extent than hemoglobin S (Cabannes, *et al.*, 1956). Although this may well be due to simply more gene flow having occurred, I think there are two other factors which must be considered. First, the much greater selection against the homozygote for hemoglobin S as compared to the homozygote for hemoglobin C would tend to eliminate incoming S genes at a greater rate; and hence one would expect the C genes to survive longer although just as many S genes had come across the Sahara. Second, *P. malariae* is much more predominant in the desert and even in Algeria has a considerable frequency, so that the advantage for hemoglobin C does not decrease as

rapidly as one proceeds north; hence this should be another factor contributing to the greater number of C genes in North Africa.

In summary, then, the distribution of hemoglobin C in some parts of West Africa accords with the hypotheses put forward in this paper, while in other areas it does not. The areas where it does not are in the extreme east (Eastern Nigeria, Southern Cameroons) and the extreme west (Senegal to Liberia). But the fact that there are areas where selective advantages of the genes do not seem to completely explain the distribution is not necessarily evidence against these hypotheses. I (Livingstone, 1958) have previously attempted to show why the sickle cell gene is not in equilibrium in many parts of West Africa, and I think one can use a similar argument for hemoglobin C; particularly since the extreme western part of West Africa where the hemoglobin C gene is not in equilibrium is also the area where the sickle cell gene is not in equilibrium. With regard to hemoglobin C the selective advantage seems to have been present for a considerable length of time in these areas, but hemoglobin C seems to be a rather recent mutation (Mourant, 1954). It is also found in the more isolated populations of West Africa, which may explain why it has not diffused more widely throughout West Africa. The highest frequencies of hemoglobin C occur among primitive sudanic agriculturalists, most of whom speak Gur languages (Sansarricq, Marill, Portier, and Cabannes, 1959; Edington and Lehmann, 1956). While the Mande peoples to the west of the Gur and the Kwa peoples to the east of them have penetrated extensively into the tropical rain forest, the Gur peoples have not. Hence the hemoglobin C gene does not seem to have been diffused at the time of the peopling of the tropical rain forest by primitive agriculturalists. On the other hand the subsequent large movements of people in West Africa which were the result of the slave trade do seem to have diffused the hemoglobin C gene to a great extent since it is found in the tropical forest in Sierra Leone, Southern Ghana, and Southwestern Nigeria, the three places in West Africa from which the majority of the slaves were exported to the New World. Thus, in addition to selection, gene flow seem to have played a considerable role in deteriming the distribution of the hemoglobin C gene in West Africa.

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A NOTE ON DIFFERENTIAL TASTE RESPONSES TO P. T. C. (PHENYL-THIO-CARBAMIDE)

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DURING the years since Fox (1932) discovered the differences in response to P. T. C. (phenyl-thio-carbamide) geneticists have continued to carry on investigations of this phenomenon. As is often the case, what appeared at the outset to be rather simple, has come to be known as more complex. While early investigators suggested that the ability to taste P. T. C. is inherited as a simple all-or-none characteristic, later evidence has shown variations in taste thresholds, and the possibility of "some differences in the penetrance of the gene in the two sexes" (Boyd, 1950, p. 279). Another factor which may add to the complexity of this problem is that of differences in *taste patterns*, or areas of the tongue responding to the P. T. C. To the best of this author's knowledge, no systematic investigation of these differences has appeared in the literature. It is hoped, therefore, that this brief note of observations made during a recent investigation will serve to stimulate further inquiry and research of a more systematic character.

MATERIALS AND METHODS

The population under study was composed entirely of white American undergraduate university students. The sample included 105 males and 105 females, 17 to 31 years old.

The subjects were tested in three groups. Each individual was given a form upon which there appeared a diagram of the tongue divided into four areas, roughly corresponding to the distribution of taste buds (Jenkins, 1954, p. 259). Two lines were drawn so as to divide the tongue, from front to back, into three sections: the tip, the blade, and the root. The blade, or central section, was then divided into equal parts by a line running from front to back.

The P. T. C. was administered on slips of paper (1.5×7.5 cm) which had been previously immersed in a 0.25% solution of P. T. C. and distilled water, and allowed to dry. The subjects were asked to place

the paper on the center of the tongue, to close the mouth, and to go through the motions of chewing. The latter procedure was designed to increase the flow of saliva, thereby distributing the chemical over the tongue. The subjects that tasted the chemical were further instructed to indicate on the diagram of the tongue those areas in which the taste was perceived. The resulting *taste patterns* were then sorted and grouped.

RESULTS

Using the above method, 72% of the group were distinguished as tasters, and 28% as non-tasters, a close approximation of the frequency characteristic of the American population as a whole. The tasters and non-tasters were nearly evenly divided between the sexes; of the tasters, 48% were male and 52% female.

Five rather distinct types, or groups of taste patterns were observed among the tasters (see table 1). Group I includes the subjects reporting

TABLE 1

Distribution of taste patterns

GROUP [1]	I	II	III	IV	V	TOTAL
Males	27	17	15	8	5	72
Females	35	17	11	11	3	77
Total	62	34	26	19	8	149

[1] See text for description of groups. $\chi^2 = 2.458$ 4d.f.
$.70 > P > .50$

distinctive taste sensations over the entire area of the tongue and, in some instances, the back and roof of the mouth. Group II includes the subjects reporting taste sensations only in the central or blade section of the tongue. The majority of this group (22) tasted the P. T. C. over all of the central section, but 9 tasted it on the left side, and three tasted it on the right side. Group III includes the subjects reporting taste sensations in the root section of the tongue. Group IV includes subjects reporting taste sensations on the tip of the tongue only. Finally, Group V, the smallest group, includes subjects who reported taste sensations on the tip and root of their tongues, but *not* in the blade or central section.

An examination of these groups with respect to the sex variable revealed no significant differences (see table 1). However, one interesting

feature suggested by these data is the possibility of a relationship between reported variations in thresholds of taste (Hartmann, 1939) and the areas of the tongue responding to the P. T. C. Moreover, there is the possibility of a relationship between reported differences in taste sensation, *i. e.*, bitter, sweet, salt, and sour (Lugg and Whyte, 1955), and the areas of the tongue responding to the P. T. C. Investigations of this phenomena will require rigorous controls inasmuch as the subjective evaluation of the individual plays such a large role.

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MIGRATION INTO THE HUMAN BREEDING POPULATION OF ANN ARBOR, MICHIGAN, 1900–1950

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INTRODUCTION

IN 1950 Lee R. Dice of the University of Michigan initiated a Study of assortative mating in the Ann Arbor community, which Study was supported by a grant from an anonymous donor. The principal objectives of the Study were to determine (a) whether people with similar traits tend to marry one another to a greater or lesser extent than would be expected by chance, (b) whether "similar" couples tend to produce more or fewer children than do "dissimilar" couples, and (c) what the effects of assortative mating and differential fertility might be on the genetic make-up of the population.

Before these major objectives could be undertaken, however, attention had to be given to the problem of migration, for this phenomenon must be considered in any analysis of genetic variability in human populations. Unfortunately, most of the previous studies of internal migration in the United States have not been very useful for purposes of genetic study. This is because movement has been classified as "migration" and "not-migration" according to whether or not it was across a political boundary, such as a state or county line. The result is that the frequent cases of movement over short distances are not represented in the statistics on migration. For our purposes it was necessary to study the rate of migration into the Ann Arbor population in a way which would not be subject to the above mentioned limitations, and we did this by defining movement of any magnitude as migration, regardless of the political boundaries that may or may not be involved. Since the statistics which we have gathered are new and since they seem to lend themselves better to genetic interpretation than do most of those which have hitherto been published, we have seen fit to present them here.

SOURCE MATERIAL

The information used in this study was obtained from the Affidavits for License to Marry (State of Michigan Form B 134) and the Certificates of Birth (Michigan Department of Health Form B213) which are on file in the Washtenaw County Court House in Ann Arbor, Michigan. We are indebted to Mrs. Luella Smith, the Clerk of Washtenaw County, for permission to study these records. The marrage license affidavits and birth certificates pertaining only to couples of which at least one member was a resident of Ann Arbor were included in the study.

The information obtained from the marriage license affidavits consists of the birthplaces and occupations of both members of the pair. From the birth certificates the birthplaces of both parents and the occupation of the father were obtained, the occupation of the mother being unfortunately not given.

DATA

The marriage license information was used to compute the great circle distance between the marriage place, namely Ann Arbor, and the birthplace of each individual. The birth certificate information was used to compute the great circle distances between the birthplace of each child, namely Ann Arbor, and the birthplaces of its parents. Our data consist, therefore, of great circle distances, designated by r, between Ann Arbor and each individual's birthplace. For the marriage license data r is the distance between the birthplace and the marriage place of a given individual, whereas for the birth certificate data r is the distance between the birthplace of an individual and the birthplace of its child.

The great circle distances were computed by spherical trigonometry from the longitudes and latitudes of the points involved. Where town or city of birth was not recorded on the affidavits but where township, county, state, or country were given, the following approximations were used in measuring the distance from Ann Arbor.

1) When the birthplace was given in terms of a township, county, and state the geographic center of the township was used as the point of measurement.

2) When the birthplace was given as a non-Michigan township without reference to county, measurement was made to the geographic center of the state.

3) When Washtenaw County, Michigan, was given as birthplace, the

point of measurement was chosen at random in Washtenaw County, using a table of random numbers.

4) When a county other than Washtenaw County, Michigan, was given with its state or its province in Canada, measurement was made to the geographic center of the county.

5) When birthplace was given as a state or province, measurement was made to the geographic center of the state or province.

6) When the birthplace was given simply as a country, measurement was made to the national capital of that country.

The data were classified by year, sex, and occupation. The ten years represented are 1900, 1910, 1920, 1930, 1940, 1942, 1944, 1946, 1948, and 1950 for the marriage license data and 1947 for the birth certificate data. Occupations were classified according to Edwards' Alphabetical Index of Occupations and Industries, 1940, into the four categories: professional, non-professional, student, and housewife.

STATISTICAL TREATMENT

The great circle distances from Ann Arbor to each individual's birthplace were used to estimate the proportion, p, of individuals whose birthplaces are less than r miles from Ann Arbor. The frequency distribution of $x = \log(r + 1)$, although usually asymmetric, is much less skewed than that of r. Accordingly, x rather than r was used in estimating p. Square root and fourth root transformations proved less effective than the logarithmic transformation in normalizing the data.

The distribution of x was found to adequately approximate the Pearson Type III probability function in the ten representative cases to which a chi-square test of goodness of fit was applied, and on this basis it was assumed that the Type III function provides a satisfactory model for interpreting all the data.

Estimates of the mean, standard deviation, and skewness of x are given in table 1 for each category into which the data were classified, except for a few categories which did not contain enough individuals to justify statistical treatment of the type needed. From these values the proportion of individuals born within r miles of Ann Arbor can be estimated from the Pearson Type III function. In obtaining these estimates we used Carver's Mathematical Statistical Tables (1950) for values of α_3 between —1.1 and +1.1 and Pearson's Tables of the Incomplete Gamma Function (1946) for other values of α_3.

TABLE 1

Estimates of the mean, standard deviation, and skewness of the transformed variate $x = \log(r + 1)$ for the various categories of year, sex, and occupation for the Marriage License Data of 1900-1950 and for the Birth Certificate Data of 1947. The data for each category are given in the order of mean, standard deviation, and skewness. For the 1947 birth data the occupations of the females pertain to the occupations of their husbands.

	1900	1910	1920	1930	1940	1942	1944	1946	1948	1950	1947 births
MALES											
	1.884	2.227	2.189	2.143	2.154	2.264	2.310	2.042	2.267	2.367	2.391
Professional	.944	.772	.806	.709	.804	.827	.868	.841	.744	.676	.578
	—.860	—1.033	—.648	—1.430	—.958	—.924	—1.012	—1.351	—1.053	.489	.189
	1.808	1.971	1.748	1.859	1.483	1.811	2.016	1.635	1.860	2.170	2.154
Non-professional	1.250	.914	1.148	1.019	1.037	.955	.944	1.091	.912	.578	.596
	—.124	—.351	—.070	—.291	—.072	—.508	—.729	—.286	—.544	.393	.210
			2.269	2.266	2.230	2.367	2.614	2.096	2.141	2.306	2.224
Student			.632	.770	.913	.644	.652	.875	.890	.611	.538
			—.758	—.463	—.755	—.264	.344	—1.055	— .782	.636	.355
FEMALES											
			1.800	1.945	2.024	2.001	2.086	1.990	2.122	1.897	2.420
Professional			.670	.794	.935	.862	.776	.946	.869	.907	.620
			—1.991	—1.078	—.813	—.887	—1.090	—.663	—.632	—.899	.170
	1.548	1.646	1.854	1.970	1.564	1.674	1.853	1.476	1.703	1.507	2.133
Non-professional	1.522	.799	.958	1.003	.980	.956	.957	1.063	1.055	1.031	.628
	—.326	—.686	—.186	—.465	—.249	—.544	—.758	—.250	—.400	—.230	.190
				2.081	2.133	2.129	2.013	2.099	1.818	1.834	2.227
Student				.903	1.002	.949	.801	.841	1.096	1.036	.528
				.323	—.546	—.466	—.732	—.344	—.227	—.313	.429

1947

	1900	1910	1920	1930	1940	1942	1944	1946	1948	1950	1947 births
	1.233	1.840	1.448	1.675	1.611	1.686	1.638	1.597	1.278	1.562	
Housewife	1.204	.965	1.020	1.117	1.073	.983	1.083	1.095	1.140	1.172	
	.582	—.541	.087	—.123	—.320	—.457	—.277	—.321	.167	—.055	
TOTAL											
	1.624	1.909	1.755	1.942	1.708	1.857	1.976	1.722	1.864	1.957	2.225
With Students	1.241	.905	1.049	.968	1.018	.948	.949	1.046	1.001	.902	.601
	.071	—.487	—.176	—.471	—.342	—.587	—.732	—.471	—.532	—.604	.211
	1.606	1.894	1.716	1.900	1.649	1.804	1.952	1.623	1.800	1.880	
Without Students	1.250	.907	1.055	.985	1.012	.954	.959	1.068	1.005	.925	
	.084	—.484	—.113	—.458	—.315	—.566	—.726	—.358	—.590	—.605	
TOTAL MALES											
	1.864	2.067	1.876	1.992	1.736	1.946	2.089	1.803	2.014	2.250	2.226
With Students	1.160	.887	1.071	.935	1.029	.929	.934	1.034	.897	.609	.586
	—.265	—.494	—.298	—.629	—.354	—.627	—.765	—.575	—.683	.530	.215
	1.837	2.046	1.812	1.925	1.653	1.881	2.055	1.691	1.940	2.216	
Without Students	1.170	.890	1.114	.960	1.025	.950	.939	1.069	.893	.606	
	—.295	—.488	—.174	—.480	—.298	—.566	—.760	—.404	—.649	.468	
TOTAL FEMALES											
	1.383	1.751	1.634	1.893	1.680	1.768	1.862	1.642	1.714	1.663	2.223
With Students	1.278	.898	1.014	1.000	1.008	.959	.952	1.052	1.075	1.042	.614
	.433	—.517	—.083	—.410	—.335	—.549	—.720	—.374	—.337	—.332	.209
	1.384	1.746	1.630	1.879	1.645	1.730	1.843	1.567	1.694	1.619	
Without Students	1.291	.902	.992	1.007	1.001	.953	.968	1.064	1.070	1.040	
	.428	—.513	—.106	—.437	—.334	—.576	—.702	—.324	—.363	—.341	

The results of the above analyses are presented in tables 2 and 3. Table 2 gives the proportion of individuals in the various sex, year, and occupational categories who were born within respectively 10, 100, and 1000 miles of Ann Arbor, and table 3 gives the estimated median distances between Ann Arbor and the birthplaces of the individuals under consideration. For the year 1947, the values given in tables 2 and 3 pertain to the birth places of parents of children who were born in Ann Arbor; for the other years they pertain to the birthplaces of individuals who were married in Ann Arbor.

RESULTS FROM ANALYSIS OF MARRIAGE LICENSE DATA

As may be seen from table 2, the great majority of individuals married in Ann Arbor were born within less than 1000 miles of Ann Arbor, and for several categories more than 30% of the individuals were born within less than 10 miles of Ann Arbor. The maximum median distance between place of birth and place of marriage for a single category is 381 miles for male students married in 1944. Almost none of the individuals in this category were born within 10 miles of Ann Arbor, and about 27% of them were born more than 1000 miles from Ann Arbor. The minimum median distance for a single category is 12 miles for housewives married in 1900, 76% of whom were born within less than 100 miles of Ann Arbor.

The most obvious regularities in the data are the differences between sexes and between occupations. Thus, except for non-professional workers in 1920, 1930, and 1940 the medians for the males exceed the corresponding values for the females. This result simply reflects the well known fact that marriages usually take place near the home of the female. However, although females tend to be less migratory than males for a given occupational group, professional and student females are more migratory than non-professional males. The differences between sexes as measured by the ratio of male to female median become progressively smaller from 1900 through 1940, after which time the sex difference again increases through 1950. The medians for professional workers are without exception greater than those for non-professional workers of the corresponding sex and year. Also, except for females in 1930, the medians for students of a given sex and year exceed those for non-professional workers.

The median distances between place of birth and place of marriage differ also in the various years, and, although considerable irregularity

TABLE 2

Proportion of individuals in the various sex, year, and occupational categories who were born within respectively 10, 100, and 1000 miles of Ann Arbor. For the year 1947 these proportions pertain to the birthplaces of the parents of children who were born in Ann Arbor; for the other years they pertain to the birthplaces of individuals who were married in Ann Arbor. For the 1947 birth data, the occupations of the females refer to the occupations of their husbands.

	1900	1910	1920	1930	1940	1942	1944	1946	1948	1950	1947 births
MALES											
	.17	.08	.09	.08	.10	.08	.09	.12	.07	.01	.01
Professional	.49	.33	.37	.34	.36	.33	.31	.39	.30	.31	.26
	.91	.86	.85	.97	.87	.81	.78	.93	.85	.83	.85
	.27	.15	.26	.21	.33	.20	.15	.28	.18	.01	.02
Non-professional	.56	.49	.58	.54	.69	.55	.45	.62	.53	.41	.41
	.83	.88	.86	.87	.93	.91	.86	.90	.91	.92	.92
			.04	.07	.10	.03	.00	.12	.11	.00	.01
Student			.30	.34	.35	.28	.17	.39	.39	.34	.36
			.90	.83	.79	.84	.73	.87	.84	.87	.92
FEMALES											
			.12	.13	.14	.13	.10	.17	.11	.16	.01
Professional			.50	.46	.44	.44	.39	.50	.41	.49	.26
			1.00	.96	.86	.90	.92	.89	.85	.92	.83
	.34	.21	.19	.17	.29	.24	.18	.33	.25	.32	.03
Non-professional	.59	.64	.55	.48	.67	.61	.51	.68	.59	.68	.43
	.82	.98	.89	.85	.94	.93	.91	.93	.90	.93	.91
				.12	.14	.13	.12	.11	.23	.21	.00
Student				.49	.42	.42	.45	.44	.55	.54	.36
				.85	.80	.82	.91	.86	.86	.87	.92
	.48	.19	.35	.28	.29	.24	.28	.29	.43	.33	
Housewife	.76	.54	.71	.61	.62	.60	.62	.63	.75	.64	
	.92	.90	.93	.88	.91	.93	.90	.91	.93	.89	

TABLE 3

Median distances in miles from Ann Arbor to birthplaces of individuals in the various sex, year, and occupational categories. For the year 1947 the medians pertain to the distance between the birthplaces of parent and offspring. For the other years they pertain to the distance between an individual's birthplace and marriage place. For the 1947 birth data the occupations of the females refer to the occupations of their husbands. The numbers in parentheses represent the numbers of individuals in each category.

	1900	1910	1920	1930	1940	1942	1944	1946	1948	1950	1947 births
MALES											
Professional	105 (21)	223 (38)	185 (39)	195 (51)	191 (93)	243 (93)	280 (73)	165 (60)	251 (74)	204 (79)	235 (251)
Non-professional	67 (61)	107 (120)	58 (229)	109 (177)	31 (247)	76 (511)	137 (471)	48 (402)	85 (306)	162 (267)	136 (564)
Students	— (5)	— (7)	223 (44)	212 (56)	222 (62)	250 (94)	381 (35)	178 (178)	179 (222)	175 (215)	154 (243)
FEMALES											
Professional	— (9)	— (16)	99 (22)	121 (52)	139 (61)	134 (102)	167 (77)	102 (76)	161 (84)	107 (117)	251 (252)
Non-professional	45 (25)	54 (66)	75 (115)	111 (139)	38 (258)	55 (399)	94 (328)	33 (329)	59 (329)	34 (247)	129 (558)
Students	— (2)	— (3)	— (12)	108 (20)	162 (30)	159 (68)	127 (65)	138 (91)	70 (97)	76 (114)	154 (243)
Housewives	12 (52)	82 (83)	26 (163)	49 (78)	45 (70)	57 (129)	48 (108)	44 (148)	17 (93)	37 (85)	— —
TOTALS											
Total Males	80 (87)	136 (165)	84 (312)	121 (284)	62 (429)	109 (698)	160 (579)	79 (640)	129 (602)	157 (561)	161 (1058)
Total Females	19 (88)	67 (168)	43 (312)	91 (289)	53 (419)	71 (698)	93 (578)	50 (644)	58 (603)	52 (563)	159 (1053)
Grand Total	40 (175)	95 (333)	60 (624)	104 (573)	58 (848)	88 (1396)	122 (1157)	63 (1284)	89 (1205)	110 (1124)	160 (2111)

exists, there is a tendency for such changes to be correlated with one another in the various sex and occupational categories. A marked overall increase in dispersal coincided with World War II. In general, there is a small but steady increase in median distance with time, the medians for the years 1900 through 1920 averaging less than those for the succeeding years. These results should be regarded with reservation, however, as it is hazardous to generalize for the period 1900 to 1940 with data taken at ten year intervals.

When the marriage information is pooled over all ten periods, the median distance from birthplace to marriage place for the various sex and occupational categories increases in this order: (1) housewives, (2) female non-professionals, (3) male non-professionals, (4) female students, (5) female professionals, (6) male professionals, and (7) male students.

RESULTS FROM ANALYSIS OF BIRTH CERTIFICATE DATA

These data were analyzed only for the year 1947, and they contain no information about the occupation of the mothers. They are nevertheless superior to the marriage license data for studying the distance between generations at corresponding points in the life cycle.

The sexual difference observed in the marriage license data is not evident here, which means that the tendency for a marriage ceremony to be performed geographically near the birthplace of the female is stronger than the tendency for a child to be born near the birthplace of its mother. The occupational differences noted in the marriage license data also hold for the birth certificate data, the professional and student categories having a larger median than the non-professionals. Although information is not available concerning the occupation of the female, it is interesting to note that the same relations which hold between median distances and occupations of males hold also for the wives of men having these occupations, indicating that mating is assortative with respect to occupational background.

Dispersal between birth and reproduction tends to be greater than that between birth and marriage, as may be seen by comparing the 1947 birth data with the 1946 and 1948 marriage data of tables 2 and 3. The median distances for parents are in general greater than those for spouses, and the proportion of parents born more than 100 miles from Ann Arbor is greater than the corresponding proportion for spouses.

USE OF MIGRATION DATA IN DEFINING AND ESTIMATING POPULATION SIZE

Migration and genetic drift are two of several factors which may change the gene frequencies of a population. The effectiveness of these two modes of change depends in part upon population size—or, more strictly, upon the size of the breeding population. It is important to obtain estimates of the size of a human population, therefore, if we are to evaluate the effects of migration and drift in gene frequencies on its genetic composition. But before population size can be estimated it must be defined, and of course there is an element of arbitrariness in any definition.

If we consider the human breeding population to be species-wide, then the size of the breeding population is the total world population of reproductive ability, and the population space is the total inhabited area of the earth. In this sense the rate of migation into the breeding population would be zero. For some problems in human genetics it may be appropriate to consider the total human population as a single breeding population (see, for example, McArthur and Penrose, 1951), but for most problems in human population genetics the meaningful population unit is small both in area and in number compared to the world as a whole. This is because the parents of the great majority of persons come from a rather limited geographic area. Consider, for example, the distance between the birthplace of an individual born in Ann Arbor in 1947 and the birthplaces of that individual's parents. The median distance between Ann Arbor and the birthplaces of parents of offspring born in Ann Arbor is equal to some fraction of the great circle circumference of the earth. If the distribution of world population were of uniform density and if the birthplaces of parents of children born in Ann Arbor were distributed at random over the earth, the median distance between Ann Arbor and the birthplaces of the parents would equal one-fourth of the great circle circumference of the earth, or roughly 6200 miles. We found that the observed median distance between Ann Arbor and the birthplaces of parents of children born in Ann Arbor in 1947 was 160 miles. Thus the median distance expected under conditions of randomness is almost 39 times the median distance estimated from our data. This finding gives quantitative support to the obvious conclusion that the parents of offspring born in Ann Arbor are not a random sample of the parents of the world, nor even the parents of the United States. Ann Arbor is to a considerable extent isolated from the population of the rest of the world, if only by distance. Of

course it has been known for some time that geographic propinquity is an important factor in mate selection (see, for example, the pioneer study of Bossard, 1932, and the confirmatory studies of Harris, 1934, Davie and Reeves, 1939, Abrams, 1943, Ellsworth, 1948, Koller, 1948, 1951, and Marches and Turbeville, 1953), but we have not previously had satisfactory measures of this spatial factor for an area larger than a city.

The preceding discussion has indicated the necessity for considering the population of local areas apart from the population of the world, but it has not suggested how a local population might be defined. In choosing a boundary to define a local breeding population within a large continuous population two opposite kinds of error can be made. If the area is too small, an appreciable proportion of the parents of children who are actually born in the local breeding population will be excluded. If the area is too large, an appreciable proportion of individuals who may be parents but who are not parents of individuals born in the local breeding population would be included within the area. An ideal definition requires a model in which these two opposite errors exactly compensate for one another. Wright (1943, 1946) has developed a model which achieves this desired result, but it does so at the expense of assuming uniform population density. The population size estimated in this way might be called the *effective* size of the population. Wright's model is not applicable to our problem, however, since the density of the human population surrounding Ann Arbor is far from uniform. We do not have a mathematical model which adequately compensates for the two types of error in a continuous population of highly uneven density, and consequently we are not able to estimate the effective size of the breeding population in the sense in which this concept is defined by Wright.

But although our data do not lend themselves to estimates of effective population size in the sense of Wright, they can be used to estimate population size in terms of another quite arbitrary definition which may at least be useful for comparative purposes. The arbitrary definition of population size for the area surrounding Ann Arbor which we suggest is the number of individuals within a radius about Ann Arbor equal to the median dispersal distance estimated for migrants into the Ann Arbor population. It should be emphasized that this definition is completely arbitrary and that it does not correspond to effective population size in Wright's sense. However, population size by this definition can be estimated from the data available, and such estimates may be useful for

investigating changes in communities from time to time and in comparing various communities with one another. Accordingly, we have applied it to Ann Arbor, using the combined marriage data for 1950.

For the year 1950, the median dispersal distance between birthplace and marriage place was 110 miles. The number of individuals living within a circle of radius 110 miles drawn about Ann Arbor was ascertained from the U. S. Bureau of the Census *County and City Data Book*, 1952, and from the Dominion Bureau of Statistics, Ninth Census of Canada, 1951, Vol. 1, 1953. From these data the total population included within a radius of 110 miles from Ann Arbor was estimated to be 6.8 million people. If we let

a = total population within area = 6.8 million

b = proportion of females in total population = .49

c = proportion of females between ages of 14-44 = .49

d = proportion of females between ages 14-44 who are married = .62

e = proportion of married females between ages 14-44 who have one or more children = .84

then one estimate of the size, N, of the breeding population is

$$N = abcde = 6.8 \times .49 \times .49 \times .62 \times .84 = .85 \text{ million.}$$

We thus obtain 850,000 as a rough approximation of the size of the breeding population surrounding Ann Arbor by the arbitrary definition of population size which we propose.

A breeding population of 850,000 is from one point of view large. Such a size, however, is small compared to the world poplation or the population of the United States and Canada in 1950. Moreover, this population has a highly intricate structure. A complex interplay of geographical, biological, social, cultural, and psychological factors operates to reduce greatly the number of potential mates for a specific individual member of the population.

SUMMARY

The dispersal between place of birth and place of marriage was analyzed for males and females of several occupational groups resident in Ann Arbor, Michigan, for each of the years 1900, 1910, 1920, 1930, 1940, 1942, 1944, 1946, 1948, and 1950. Males were more migratory than females, and students and professional workers were more migratory than non-professional workers. Both sexes and all occupations were more mobile in certain years than in others.

The dispersal between birthplaces of parents and birthplaces of their children was also analyzed, though only for the year 1947. Males were not appreciably different from females in this respect, although students and professional workers were more migratory than non-professional workers. The dispersal between place of birth and place of reproduction tends to be greater than that between place of birth and place of marriage.

The area occupied by the breeding population surrounding Ann Arbor is defined to be the area of a circle circumscribed around Ann Arbor whose radius is equal to the median distance between place of birth and place of marriage. The median distance for the total 1950 series is 110 miles. Using this distance, and neglecting factors other than isolation by distance, it is estimated that 850,000 mates were available for a reproductive individual selected at random from the residents of Ann Arbor in 1950. This estimate for the area and number of the breeding isolate including Ann Arbor, although easily ascertained, differs mathematically from the theoretically more desirable but computationally more difficult panmictic unit as described by Wright (1943, 1946). Our estimate should be considered as a very rough approximation to the size of the breeding isolate within a quasi-continuous human population of variable density.

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DISTRIBUTION AND INHERITANCE OF MIDDLE PHALANGEAL HAIR IN A WHITE POPULATION OF SÃO PAULO, BRAZIL

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SINCE 1921, when Danforth suggested that the presence of middle phalangeal hair is hereditary, the results of many investigations have shown that the character presents striking racial variation. The frequency of individuals without middle phalangeal hair in different populations varies from 21.6% to 98.0% (*cf.* table 1). In the families investigated, the absence of middle phalangeal hair seems to be recessive (Bernstein and Burks, 1942; Bernstein, 1949). However, the findings of exceptions to this rule (Chopra, 1953; Matsunaga, 1956; Beckman and Böök, 1959) and differences due to age (Büchi, 1957) and sex (Garn, 1951a; see also table 1) indicate that new investigations are needed

The purpose of the present paper is to report some population and sibling data.

POPULATION DATA

During an investigation of the genetical effects of immigration in a white population of the state of São Paulo, Brazil (Saldanha, 1959), the presence or absence of middle phalangeal hair among 289 individuals, 131 males and 158 females, was determined by means of a pocket lens. The mean ages of the individuals investigated were 16.2 for males and 15.6 for females. In the examination of the fingers for middle phalangeal hair of both hands, the parental origin of the individuals was also recorded.

National origin. Since a large number of European immigrants arrived in the community investigated about two generations ago, individuals were classified according to the nationalty of their ascendants in four groups: (a) descendants of Brazilians; (b) descendants of Italians, the largest immigrant stock; (c) descendants of Brazilians and Italians (mixed); and (d) descendants, "pure" or mixed, of immigrants of other nationalities, mainly Syrian, Austrian, Swiss, German and French (remainder group).

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TABLE 1

Incidence of individuals without middle phalangeal hair in different populations

SAMPLE	LOCATION	MALES		FEMALES		BOTH SEXES		AUTHOR
		N	%	N	%	N	%	
Whites (mostly of north European derivation)	U. S. A.	—	—	—	—	111	21.6	Bernstein and Burks, 1942
Dutch *	São Paulo, Brazil	96	16.7	95	31.2	191	24.1	Saldanha *et al.*, 1960
Whites (general population)	Baltimore, U. S. A.	—	—	—	—	541	27.0	Glass *et al.*, 1952
Spaniards	Madrid	601	25.0	—	—	—	—	Valls, 1960
Swedish	Sweden	100	30.0	100	28.0	200	29.0	Beckman and Böök, 1958
Basques	Guipuzcoa, Spain	167	31.7	—	—	—	—	Valls, 1960
	San Sebastian	25	36.0	53	26.4	78	29.5	Boyd and Boyd, 1937
Germans	Germany	253	28.5	244	32.8	497	30.2	Matsunaga, 1956
Whites (general population)	Boston, U. S. A.	239	33.1	—	—	—	—	Garn, 1951b
Jews	Baghdad	69	29.0	136	40.4	205	36.6	Boyd and Boyd, 1941a
Syrians *	Boarij and Meshgara	287	33.8	143	45.4	430	37.7	Boyd and Boyd, 1941b
Armenians	Beirouth and Ghazir	165	37.6	172	38.4	337	38.0	Boyd and Boyd, 1941b
Arabs *	Baghdad	281	33.1	146	48.6	427	38.4	Boyd and Boyd, 1941a

Whites (mostly of Irish derivation)	U. S. A.	—	—	—	—	38	44.7	Bernstein and Burks, 1942
Whites (mostly of Italian derivation)	U. S. A.	—	—	—	—	58	44.8	Bernstein and Burks, 1942
Aleuts *	Aleutian Islands	42	33.3	36	58.3	78	44.9	Garn, 1951b
Whites (general population) *	U. S. A.	180	38.9	193	54.0	373	46.6	Danforth, 1921
Whites (mostly of Italian derivation) *	São Paulo, Brazil	131	40.5	158	54.4	289	48.1	Present paper
Russians *	Moscow, Tiflis and Kharkov	355	44.5	422	55.7	777	50.6	Boyd and Boyd, 1937
Hindus (Bengalis)	India	427	46.4	—	—	—	—	Büchi, 1954
Egyptians *	Cairo and Assiut	535	47.7	523	56.6	1058	52.1	Boyd and Boyd, 1937
Whites (" Dunker " Isolate)	Baltimore, U. S. A.	—	—	—	—	234	52.5	Glass *et al.*, 1952
Hindus (Nokte Naga)	Assam, India	111	54.1	—	—	—	—	Kumar, 1955
Japanese	Japan	503	55.7	494	61.1	997	58.3	Matsunaga, 1956
Hindus	India	—	—	—	—	250	75.2	Chopra, 1953
Indians (Shinnecock and Penobscot)	U. S. A.	83	72.3	67	79.1	150	75.3	Danforth, 1921
Negroes	U. S. A.	74	83.7	103	90.3	177	87.6	Danforth, 1921
Eskimos	Labrador and Baffin Islands	—	—	—	—	201	98.0	Sewall, 1939

N = sample size. * = significant sex difference.

Table 2 presents the distribution of the number of fingers with middle phalangeal hair among the four groups. The average number of fingers with hair is higher among descendants of Brazilians but this difference is significant only when Brazilian, Italian, or mixed group is compared with the remainder group. The frequency of individuals without middle phalangeal hair is not significantly heterogeneous among the four groups (table 2). However, the low frequency found among the (a), (b) and (c) groups, that can be considered as Mediterranean because of the Portuguese origin of Brazilian whites, differs significantly from the remainder

TABLE 2

Distribution of middle phalangeal hair of 289 individuals according to their parental origin

GROUP	NO. OF INDIVIDUALS	NO. OF FINGERS WITH PHALANGEAL HAIR						INDIVIDUALS WITHOUT PHALANGEAL HAIR (%)	HETEROGENEITY
		0	1	2	3	4	MEAN ± s. e.		
Desc. Brazilian	57	26	8	9	8	6	1.30 ± 0.19	45.6	$\chi^2 = 5.17$
Desc. Italian	91	38	21	16	12	4	1.15 ± 0.13	41.8	d.f. = 3
Mixed group	70	33	12	12	9	4	1.13 ± 0.15	47.1	P = 0.15
Remainder group	71	42	13	5	7	4	0.85 ± 0.15	59.2	
Total	289	139	54	42	36	18	1.10 ± 0.08	48.1	

group that mainly includes immigrants from Central Europe ($\chi^2 = 4.61$; d.f. = 1; P = 0.03). The total frequency of individuals without middle phalangeal hair is similar to that found among whites in the United States (44.8%), mostly of Italian derivation (Bernstein and Burks, 1942).

Sex and age differences. From the examination of table 1, it appears that there may be sex differences in the frequency of individuals with middle phalangeal hair. However, these differences are not evident in every population studied. This situation suggests that sampling heterogeneity should account for a large part of sex differences. Table 3 shows that sex difference in the frequency of middle phalangeal hair is highly significant and sampling heterogeneity in the present study is practically absent. In spite of this result it can be seen in table 1 that only 8 out of 16 investigations in which sex was recorded showed a significant sex difference. Most of these findings cannot be due to

sampling heterogeneity. Sum of χ^2 values obtained for 16 samples in table 1 was 68.37 (d.f. = 16, $P < 0.0001$) and pooled χ^2 was 54.73 (d.f. = 1, $P < 0.0001$), so that the difference between these figures is not significant ($\chi^2 = 13.64$, d.f. = 15, $P = 0.65$).

Similarly, age differences have been detected in investigations of middle phalangeal hair (Büchi, 1957). It is interesting that such

TABLE 3

Comparisons of frequencies of individuals with middle phalangeal hair between males and females, according to their origin

GROUP	SEX	TOTAL	INDIVIDUALS WITH PHALANGEAL HAIR NO.	%	χ^2 (*)	P
Brazilian descendants	Male	27	17	63.0	1.52	= 0.20
	Female	30	14	46.7		
Italian descendants	Male	43	29	67.4	2.84	= 0.10
	Female	48	24	50.0		
Mixed Group	Male	27	17	63.0	1.80	= 0.17
	Female	43	20	46.5		
Remainder Group	Male	34	15	44.1	0.29	= 0.60
	Female	37	14	37.8		
Total	Male	131	78	59.5	5.60	= 0.02
	Female	158	72	45.6		

* Sum of four χ^2 values = 6.45, d.f = 4, $P = 0.17$.
Difference (heterogeneity) between sum and pooled χ^2 values = 0.85, d.f. = 3, $P = 0.85$.

differences are greater among males than among females. Since Garn (1951b) showed that the frequency of individuals with middle phalangeal hair among castrated males is lower than among controls from the general population, hormonal differences to be found among individuals of different ages might reflect a variation of phalangeal pilosity with aging. To test this hypothesis table 4 presents the distribution of number of fingers and frequency of individuals with middle phalangeal hair by the development of puberty. The beginning of puberty was taken at 15 for males and 13 for females. The average number of fingers and the frequency of individuals with phalangeal pilosity for both males and females are higher after the beginning of puberty. The increase

TABLE 4

Sex distribution of the numbers of fingers and individuals with middle phalangeal hair at periods before and after the beginning of puberty

SEX	AGE GROUP	TOTAL	NO. OF FINGERS WITH HAIR					INDIV. WITH PHALANGEAL HAIR		χ^2 (2)	AVERAGE NO. OF FINGERS WITH HAIR	t
			0	1	2	3	4	NO.	%			
Males	before puberty	65	32	11	13	6	3	33	50.8	4.12	1.03 ± 0.15	2.42
(1)	after puberty	66	21	15	11	13	6	45	68.2	$P = 0.04$	1.52 ± 0.17	$P = 0.007$
	all ages	131	53	26	24	19	9	78	59.5 ± 4.3		1.27 ± 0.11	
Female	before puberty	45	26	11	3	5	0	19	42.2	0.28	0.71 ± 0.15	1.86
(1)	after puberty	113	60	17	15	12	9	53	46.9	$P = 0.65$	1.05 ± 0.13	$P = 0.06$
	all ages	158	86	28	18	17	9	72	45.6 ± 4.0		0.96 ± 0.10	
Both sexes	before puberty	110	58	22	16	11	3	52	47.3	1.53	0.90 ± 0.11	2.41
	after puberty	179	81	32	26	25	15	98	54.8	$P = 0.20$	1.22 ± 0.10	$P = 0.007$
	all ages	289	139	54	42	36	18	150	51.9 ± 2.9		1.10 ± 0.08	

(1) The beginning of puberty was considered as 15 years for males and 13 years for females.
(2) Sum of χ^2 for both sexes = 4.40, d.f. = 2, $P = 0.11$; heterogeneity, $\chi^2 = 2.87$, d.f. = 1, $P = 0.09$.

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sex	Males	Females	Both sexes %
Di			
wi di la			
si I f th I r n			
M			

of phalangeal pilosity with the development of puberty for males is significantly marked. This fact suggests that the pilosity on the middle phalanges is influenced by hormonal changes. Since these changes are appreciably more pronounced among males, probably they are due to the variation of androgens with sex and age.

The frequency of individuals of both sexes with middle phalangeal hair before and after the beginning of puberty is not statistically significant ($\chi^2 = 1.53$, d.f. $= 1$, $P = 0.20$). Since difference in average number of fingers with phalangeal hair between sexes was found ($t = 2.41$, $P = 0.007$), sampling heterogeneity ($\chi^2 = 2.87$, d.f. $= 1$, $P = 0.09$) in the frequency of individuals with middle phalangeal hair could be due to different changes of androgen rates among males and females.

Other factors. The distribution of the combination of the fingers

TABLE 5

Distribution of combinations of fingers with middle phalangeal hair (left hand)

SEX	TOTAL	FINGER WITH PHALANGEAL HAIR								
		NONE	IV	III	II	V	III+IV	IV+V	III+IV+V	II+III+IV+V
Males	131	63	15	3	2	0	21	2	19	6
Females	158	93	25	2	1	1	16	0	12	8
Both No.	289	156	40	5	3	1	37	2	31	14
sexes %	—	54.0	13.8	1.7	1.0	0.4	12.8	0.7	10.7	4.9

with middle phalangeal hair does not seem to occur at random. This distribution shows a pattern similar to those observed in other populations (Danforth, 1921; Bernstein and Burks, 1942; Garn, 1951a; Matsunaga, 1956; Beckman and Böök, 1959).

In both sexes the presence of middle phalangeal hair in fingers considered individually has the following relative frequency order: IV → III → V → II fingers (table 5). In spite of the variation in relative frequency of combinations of types of fingers, the frequency order of the fingers with middle phalangeal hair is the same in different populations. These findings suggest a morphogenetic bilateral gradient from IV finger. Bernstein (1949) has presented data which suggest that the range of the embryonic areas for middle phalangeal hair might be determined by different allelic genes.

(2) Sum of χ^2 for both sexes = 4.40, d.f. = 2, $P = 0.11$; heterogeneity, $\chi^2 = 2.87$, d.f. = 1, $P = 0.09$.

Differences between left and right hands present another set of problems. The distribution of fingers with middle phalangeal hair is not exactly the same for both hands. Table 6 shows the distribution found in the present sample. The difference in distribution of phalangeal pilosity between right and left hands is small. When both hands are considered, the frequency of individuals without middle phalangeal hair decreases. The importance of these differences which should be due to constitutional and environmental factors can be evaluated on statistical

TABLE 6

Distribution of the number of fingers with middle phalangeal hair in right and left hands

HAND CONSIDERED		NUMBER OF FINGERS WITH MIDDLE PHALANGEAL HAIR					
		0	1	2	3	4	Total
Right	no.	152	50	39	40	8	289
	%	52.6	17.3	13.5	13.8	2.8	—
Left	no.	156	49	39	31	14	289
	%	54.0	17.0	13.5	10.7	4.8	—
Both hands	no.	139	54	42	36	18	289
	%	48.1	18.7	14.5	12.5	6.2	—

grounds. The average number of fingers with phalangeal pilosity in left hands (0.955 ± 0.073) is lower than that in right hands (0.969 ± 0.071) but the difference is not significant ($t = 1.40$, $P = 0.20$). The coefficient of correlation between the number of fingers with middle phalangeal hair in both hands is $+0.893 \pm 0.012$, being highly significant ($z = 1.436 \pm 0.059$; $z/s.e. = 24.3$, $P < 0.001$). However, distribution of fingers with phalangeal hair is clearly skewed.

Moreover, the frequency distribution of individuals according to the four types of combinations between right and left hands with phalangeal hair is:

Middle phalangeal hair present on				
both hands	*left hand*	*right hand*	*no hands*	*total*
120	17	13	139	289

This distribution shows that the association between right and left hands (Yule's coefficient of association, $Q = 0.974 \pm 0.010$) is highly

significant ($\chi^2 = 181.20$, d.f. $= 1$, $P < 0.0001$). Table 7 shows that the number of fingers with middle phalangeal hair on the right is lower than that on the left hand. However, the frequency of asymmetries between both hands should not be important in population investigations. From table 7, it can be seen that the frequency of asymmetrical combinations among females is lower than among males. This also may be caused by hormonal differences.

TABLE 7

Frequencies of assymmetrical combinations between fingers with middle phalangeal hair of right (R) and left (L) hands

SEX	NO. OF INDIVIDUALS	SYMMETRICAL COMBINATION		ASSYMMETRICAL COMBINATION: LESS NO. OF FINGERS WITH HAIR ON R.		LESS NO. OF FINGERS WITH HAIR ON L.		ATYPICAL COMBINATION	
		no.	%	no.	%	no.	%	no.	%
Males	131	91	69.5	13	9.9	21	16.0	6	4.6
Females	158	125	79.1	15	9.5	15	9.5	3	1.9
Both sexes	289	216	74.7	28	9.7	36	12.5	9	3.1

SIB-PAIR DATA

The character was also observed in 47 sib pairs, collected at random from the same population. The sib-pair group includes 40 males and 54 females from 10 to 20 years old. In the present analysis only the the presence or absence of middle phalangeal hair is considered.

On the grounds of family observations, Danforth (1921) suggested that the absence of middle phalangeal hair might be determined by recessive genes. Genetical analysis of sibling and family data (Bernstein and Burks, 1942; Bernstein, 1949) seems to confirm this hypothesis. However, it should by no means be accepted as a too rigid scheme since 69 Japanese matings of parents without middle phalangeal hair produced 8 children with phalangeal pilosity (Matsunaga, 1956). Similarly, a single exception was observed among Hindu (Chopra, 1953) and Swedish (Beckman and Böök, 1959) family data.

Cotterman (1937) developed a method for monogenic analysis based on sib-pair type distribution. To apply Cotterman's single generation method, presence (A) or absence (B) of middle phalangeal hair was

postulated as determined by allelic genes, frequencies of which in the population are p and $(1-p)$, respectively. Assuming the character A to be dominant over B, the expected proportions of sib-pair types would be:

Both sibs with the character A (AA)
$$- f_{aa} = \tfrac{1}{4}p(4+5p-6p^2+p^3).n$$

One sib with the character A and other with B (AB) $- f_{ab} = \tfrac{1}{2}p(1-p)^2(4-p).n$

Both sibs with the character B (BB)
$$- f_{bb} = \tfrac{1}{4}(1-p)^2(2-p)^2.n$$

where n stands for the number of sib pairs in the sample.

The most convenient estimate of p is obtained from the proportion of individuals with the recessive character ($\bar{B}$) among the sib-pair group:

$$p = 1 - \sqrt{\bar{B}}$$

The alternative random combination hypothesis should also be tested. Assuming sib combinations as random, the expected frequencies of sib-pair types is obtained by means of the binomial rule:

$$(\bar{A}+\bar{B})^2.n = (\bar{A}^2+2\overline{AB}+\bar{B}^2).n$$

Table 8 shows the observed and expected frequencies of sib-pair types,

TABLE 8

Frequencies of types of sib pairs observed and expected from monogenic recessivity and random combination hypothesis (A indicates presence and B absence of middle phalangeal hair in both hands)

TYPE OF SIB-PAIR COMBINATION	OBSERVED FREQUENCY	EXPECTED FREQUENCY: RECESSIVITY HYPOTHESIS	χ^2 P (d.f. = 1)	RANDOM COMBINATION HYPOTHESIS	χ^2 P (d.f. = 2)
AA	11	13.8	3.61; > 0.05	47.0	2.20; = 0.35
AB	18	12.3	0.57	8.6	0.67
BB	18	20.9	2.64	23.0	1.09
Total	47	47.0	0.40	15.4	0.44

A = 42.6% B = 57.4% p = 0.242

on the alternative hypothesis of monogenic recessivity and random combination. The expected values for both hypotheses do not differ from the observed ones, at 5% level of probability. However, the observed value for AB combination in the case of monogenic recessivity is in excess. Since, in the sample, 26 sib pairs are heterosexual and 21 include sibs in pre- and post-puberty periods, departure from expected ratio considering age and sex differences analysed in table 3, need not be in disagreement with a main pair of allelic genes. Similarly, the exceptions among the Japanese, Swedish and Hindu family data could be caused by differences to be found especially between two different generations.

SUMMARY

From the present analysis and discussion it seems to be permissible to draw the following conclusions:

1. The frequencies of individuals without middle phalangeal hair show striking population differences. The range among North Europeans varies from 20 to 30%; among the Mediterranean, from 30 to 50%. Among Japanese, American Indians and Negroes, the figures vary between about 60 and 90%. The trait is virtually absent among Eskimos.

2. The frequency of individuals without middle phalangeal hair presents sex and age differences. Sex differences found in samples from different populations show great heterogeneity which could be a reflection from other factors such as sampling, environmental conditions, and, mainly, age distribution. However, the variation of male hormone rates with age and sex should be counted as the main factor in determining intrapopulation heterogeneity.

3. The frequencies of fingers without middle phalangeal hair among right and left hands are slightly different. Since right hands present a higher frequency of fingers with phalangeal pilosity, this does not seem to be caused by environmental factors. However, because of its smallness, this difference may be discarded in population studies.

4. The frequency order of the types of fingers with middle phalangeal hair: IV → III → V → II fingers seems to be universal. It is possible that such a morphogenetic gradient is under the action of different allelic genes with variable frequencies among different ethnic groups.

5. The lack of middle phalangeal hair, in its fundamental scheme, may be determined by a pair of recessive genes. However, the occurrence of sex, age, and possibly environmental differences make difficult the

genetical analysis of the trait, mainly in data from different generations (family data).

6. From the above, it is of interest in the collection of future population, sibling, and family data that age and sex be recorded in order to make possible a more accurate population and genetical analysis. While the presence of middle phalangeal hair is a character of anthropological importance, it is not ideal for genetical investigations.

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AGE AT MENARCHE IN SOUTH AFRICAN BANTU SCHOOLGIRLS LIVING IN THE TRANSKEI RESERVE[1]

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THE age at which the first menstrual period, or menarche, occurs is a measure much used by students of growth and development in assessing the speed of maturation of individuals, or, on a group basis, of populations. It is known to be influenced by both hereditary and environmental factors. State of nutrition is the most important environmental factor; climate was formerly thought to exert some effect, but modern data make it clear that its influence, if any, is very limited. Within a relatively homogeneous culture such as that represented by the South of England or by Denmark, the children of the top socio-economic groups reach menarche two or three months earlier than children of the lowest socio-economic groups. There is some evidence of racial differences, or differences associated with national origin, but the over-riding effects of nutrition have obscured any certain knowledge of these. Currently the average age of menarche in school children in London and the South of England is 13.1 years; the average for a well-nourished, well-circumstanced group of Burmese and Assamese has been reported as 13.2 years (for bibliography see Tanner, 1961).

In these circumstances extensive data from different population groups, and particularly non-European ones, are to be welcomed, and this paper reports statistics for a sample of 47,420 South African Bantu girls living in the Transkei reserve and divided into two groups on a socio-economic basis. The paper is of additional interest, we hope, in that for the first time a test of the logistic distribution, as well as the more commonly used normal distribution, has been made for fitting the proportions of girls having menstruated at each month of age. It

[1] This survey was undertaken at the suggestion of Dr. A. G. Oettlé of the South African Institute of Medical Research. We are most grateful to him for this and for his help in the planning of the investigation.

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requires a very large sample to distinguish the relative merits of these two very similar distributions.

MATERIAL AND METHODS

The population studied consisted of approximately 50,000 Bantu school girls living in the Transkeian territories, a Bantu reserve in the Union of South Africa with a relatively homogeneous population of about 1.6 million. The survey took place during the four days 4th-7th August, 1958, that is, in early spring. Through the courtesy of the regional Director of Bantu education questionnaire forms were distributed to all schools in the territory and 92% of these forms were returned complete with information on whether menarche had occurred or not, and date of birth. In all 47,420 girls returned usable papers.

School girls aged 10–18 years (specifically 120–215 months since birth) were interviewed individually in private by Bantu woman teachers who enquired whether menstruation had yet occurred. Bantu girls experience no embarrassment at answering this question; they are proud of reaching maturity.

Thereafter the Bantu school principal, assisted by the girls' class teacher, assessed whether the home conditions were economically "poor" or "not poor." The use of more objective economic criteria had been discussed previously at a congress of Bantu school authorities and on balance it was thought that the simple dichotomous rating would be at least as serviceable as any more sophisticated device. The category "not poor" does in fact imply very much the same circumstances throughout the whole territory we investigated, as the category "poor " does also.

The date of birth was ascertained from the school admission register. When a child is brought to school for the first time the mother is questioned meticulously by the school principal about its month and year of age. Children are not admitted until they are seven years old and this education department directive is rigidly enforced. The time of birth is first fixed by reference to the dates of well-known events in the vicinity and then the mother is asked in which of the four seasons she gave birth to the child. She will reply that the child was born early mid or late in one of these seasons. The principal will then narrow down to the month, all of which have Xhosa names which are accepted and used throughout the whole territory. Thus, though many fully grown school girls have only the vaguest idea of how old they are, the school admission register is to be regarded as highly reliable. Discussion

with school inspectors, both white and Bantu, and actual observation of school principals making the admission enquiries of the mothers reinforces our opinion that these ages are nearly all correct to the nearest month.

The sample was divided into two groups; 27,036 girls from "poor" homes and 20,384 girls from "not poor" homes. For each group a table was made giving the number and percentage of girls who had experienced menarche for each month of age from 120 months (nil) to 215 months (98% in poor, 100% in not poor). The totals in each monthly group varied from 493 down to 75.

When the proportion of girls menstruating is plotted against age, a fairly symmetrical sigmoid curve is produced. Two mathematical models are in common use for this type of data; the cumulative Normal curve,

$$\hat{p} = \int_{-\infty}^{\beta(x-\mu)} \exp(-\tfrac{1}{2}u^2)\,du$$

and the logistic curve,

$$\hat{p} = (1 + \exp(-\beta(x-\mu))^{-1}$$

where in each case $\hat{p}$ is the expected proportion menstruating, x the age, and μ and β numerical constants, μ representing the age at which $\hat{p} = 0.5$ and β providing an (inverse) measure of the variability of the age at menarche. These two curves are very alike except for proportions near 0 and 1, the logistic having longer " tails," and the present body of data is one of the very few known to us which is sufficiently extensive to distinguish between them.

The cumulative Normal and the logistic curves can be converted into straight lines by transforming the percentages into probits or logits respectively, and the constants μ and β can be estimated by maximum likelihood (Finney, 1952; Berkson, 1957).

RESULTS

Inspection of the data disclosed several abrupt changes in the observed proportions menstruating, occurring at twelve-monthly intervals. This suggested a possible seasonal effect, and to investigate this further the figures were treated in twelve sub-groups according to the month of birth. The results of fitting Normal and logistic sigmoids

are summarised in table 1, which gives the estimates m and b of the parameters and values of χ^2 measuring the goodness of fit of the curves.

The data for the not-poor groups are well fitted by both the Normal and logistic curves. One month gives a significantly bad fit ($P < 0.01$) to the Normal model; the logistic gives the smaller χ^2 for ten out of

TABLE 1

Estimates of parameters and goodness of fit tests for the normal and logistic models

Month of birth	Total no.	Normal			Logistic		
		m (years)	b	χ^2(6 d.f.)	m (years)	b	χ^2(6 d.f.)
				Poor group			
Jan.	2013	15.57	0.64	27.94	15.58	1.18	10.43
Feb.	1998	15.59	0.66	45.53	15.60	1.22	7.97
Mar.	2583	15.65	0.63	11.14	15.64	1.16	0.57
Apr.	2356	15.54	0.67	14.06	15.53	1.21	5.34
May	2541	15.48	0.65	44.54	15.47	1.20	6.62
June	2777	15.40	0.66	46.16	15.39	1.21	6.45
July	2406	15.37	0.67	5.12	15.36	1.21	3.05
Aug.	2183	15.35	0.68	3.63	15.35	1.21	3.89
Sep.	2209	15.33	0.69	13.89	15.34	1.24	7.18
Oct.	1945	15.28	0.71	19.70	15.29	1.28	5.66
Nov.	1939	15.24	0.68	23.79	15.25	1.24	4.34
Dec.	2086	15.24	0.63	6.51	15.25	1.12	2.07
				262.01 (72 d.f.)			63.57 (72 d.f.)
				Not-poor group			
Jan.	1538	15.08	0.80	4.16	15.08	1.44	2.50
Feb.	1510	15.13	0.78	6.17	15.14	1.40	4.77
Mar.	1979	15.13	0.79	17.12	15.14	1.43	9.06
Apr.	1809	15.15	0.78	11.67	15.16	1.40	9.29
May	1870	15.06	0.78	2.00	15.05	1.40	1.87
June	1864	15.07	0.74	4.01	15.07	1.33	3.33
July	1819	15.01	0.76	6.76	15.02	1.38	0.96
Aug.	1738	15.03	0.75	2.55	15.03	1.34	2.27
Sep.	1660	15.00	0.78	3.88	15.01	1.40	4.70
Oct.	1463	14.89	0.84	5.43	14.89	1.51	5.15
Nov.	1535	14.84	0.80	10.70	14.84	1.42	7.95
Dec.	1599	14.83	0.73	3.09	14.83	1.28	6.26
				77.54 (72 d.f.)			58.11 (72 d.f.)

the twelve months. The data for the poor group are again well fitted by the logistic model, but the Normal curve gives notably bad fits to more than half the monthly sub-groups. The nature of the fit is illustrated in figs. 1-4, where the whole of the data, grouped by six-monthly intervals for clarity, is plotted using both the probit and logit trans-

formations. For the poor group, the Normal model gives expected proportions that are markedly too low at the early ages. The logistic model will be used for the rest of this paper. It will be noted that the two models give practically identical results for the estimates of μ, the mean age at menarche.

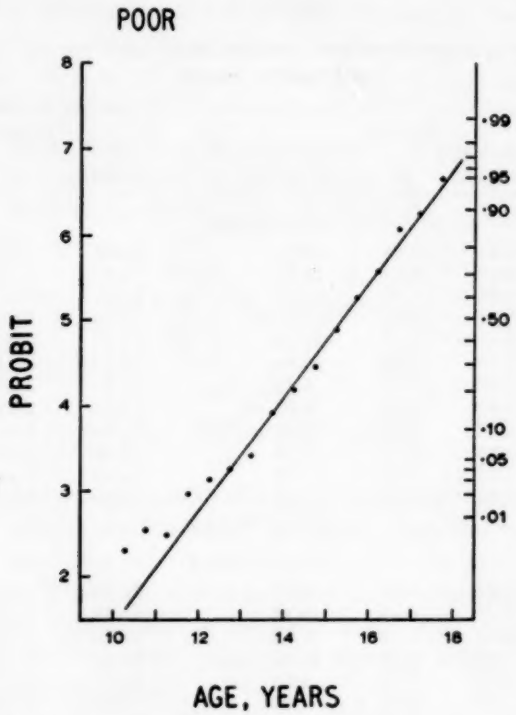

FIG. 1. FREQUENCIES OF SOUTH AFRICAN BANTU SCHOOLGIRLS FROM POOR HOMES WHO HAVE REACHED MENARCHE, PROBIT TRANSFORMATION.

The standard errors of the estimates b are approximately ± 0.055 for the poor group and ± 0.071 for the not-poor group. It follows that the twelve logit lines in each group may be considered parallel, the appropriate values of χ^2, with 11 d.f., being 6.05 and 8.06 respectively. The lines for the poor group are decidedly flatter than those for the not-poor group, the mean slopes being 1.208 and 1.395 with a difference of 0.187 ± 0.026. This implies that the ages in the poor group are less

concentrated about their mean value than are those in the not-poor group (see table 3).

The values of m, which estimate the mean age at menarche, show a pronounced seasonal trend in both the economic groups (figure 5). The mean age is greatest for girls born in the summer months January-

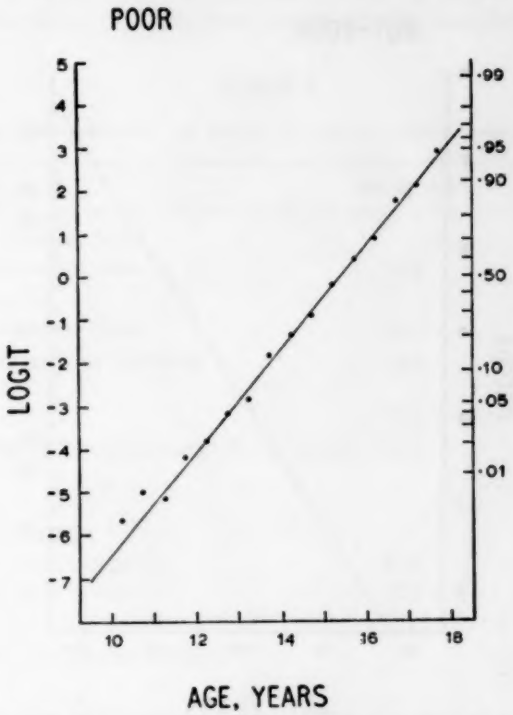

FIG. 2. FREQUENCIES OF SOUTH AFRICAN BANTU SCHOOLGIRLS FROM POOR HOMES WHO HAVE REACHED MENARCHE, LOGIT TRANSFORMATION.

March, and declines steadily over the rest of the year; there appears to be an abrupt increase of 3–4 months between girls born in December and those born in January. The mean age over all the sub-groups is 15.42 $\pm$ 0.04 years for the poor group and 15.02 $\pm$ 0.03 years for the not-poor group (the standard errors have been derived somewhat arbitrarily from the variation between the monthy sub-groups); the differences range from 0.50 years to 0.32 years with standard errors of $\pm$ 0.07 years and some indication of a minimum in summer and a maximum in winter.

As a by-product of the investigation, table 1 and figure 5 include the total numbers in the monthly sub-groups. It will be seen that these vary quite widely with the largest values corresponding to birthdays in March-June (autumn and winter).

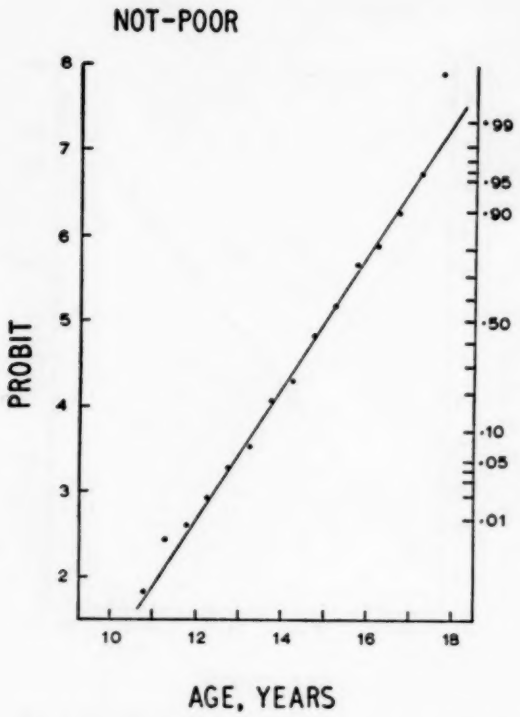

FIG. 3. FREQUENCIES OF SOUTH AFRICAN BANTU SCHOOLGIRLS FROM NOT-POOR HOMES WHO HAVE REACHED MENARCHE, PROBIT TRANSFORMATION.

DISCUSSION

As might be anticipated, the mean age at menarche of even the not-poor group of Bantu girls is high compared to all groups previously studied. For comparison, the mean ages for some other late maturing groups are given in table 2. These are all based on fairly recent studies;

it must be remembered that, in highly developed countries of the West at least, the mean age at menarche has been decreasing steadily for at least a century at a rate of $\frac{1}{3}$ year per decade. In 1959, the mean age for London schoolgirls was 13.1 years. Besides being the latest maturing, the Bantu are probably among the worst off groups economically of those so far studied. Any genetical influences tending to produce earlier or later menarche than in Europeans are at present completely masked.

TABLE 2

Mean age at menarche (in years) in certain late-maturing groups

GROUP STUDIED	MEAN AGE	AUTHORITY [1]
Durban, S. A.—Indians from lowest socio-economic class	13.9	Kark
Nigerian schoolgirls, upper socio-economic class	14.3	Ellis
Ceylon, rural areas near Columbo	14.4	Wilson & Sutherland
Eskimo	14.4	Levine
Alexandra Township (Johannesburg) S. A.—Bantu	14.9	Oettlé & Higginson (1961)
Transkei, S. A.—Bantu		
not-poor group	15.0	present study
poor group	15.4	

[1] Full citations in Tanner (1961).

The five month difference in mean age at menarche between the poor and not-poor groups confirms earlier findings. It is also not unexpected to find the poor group with a smaller logit slope, implying a wider range of ages at menarche. The standard deviations of age at menarche, as indicated by the probit fits, are 1.5 years for the poor group and 1.3 years for the not-poor group. These are probably the best figures for comparison with other studies, although the true standard deviations given by the logistic model are considerably higher owing to the larger number of extreme ages. The comparable figure for London schoolgirls in 1959 is 1.1 years. There is some tendency for a high standard deviation to be associated with a high mean age, but the relation is not a close one; thus Foll (1961) reports two series from Burma and

Assam, both with mean ages of 13.2 years, but with standard deviations of 1.1 and 1.6 years, respectively.

The larger mean and standard deviation in the poor group result in the proportion menstruating very early being almost the same in the two economic groups of our data. This is illustrated in table 3 which

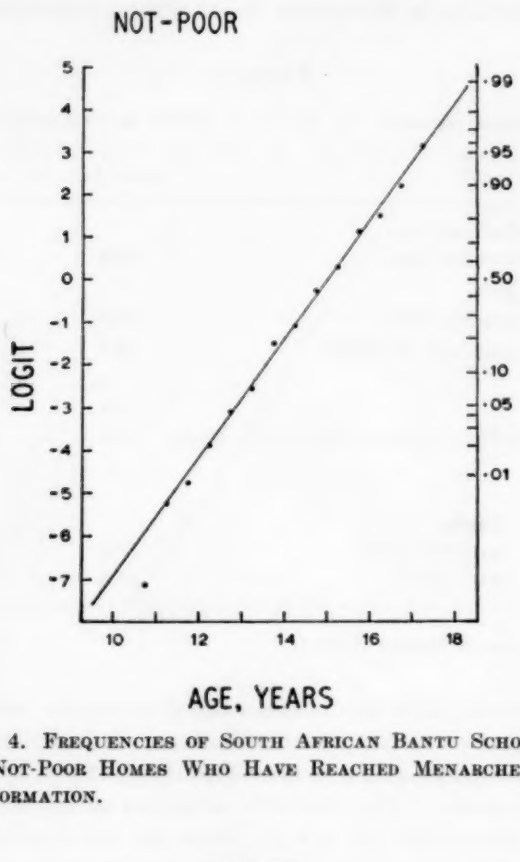

FIG. 4. FREQUENCIES OF SOUTH AFRICAN BANTU SCHOOLGIRLS FROM NOT-POOR HOMES WHO HAVE REACHED MENARCHE, LOGIT TRANSFORMATION.

TABLE 3

Percentiles of the distributions of age at menarche

PERCENT MENSTRUATING	10	25	50	75	90
Age (years)—poor	13.6	14.5	15.4	16.3	17.2
not-poor	13.4	14.2	15.0	15.8	16.6

gives the expected ages at which the proportion menstruating takes the values 0.10, 0.25, 0.50, 0.75 and 0.90 respectively. The relatively high proportions menstruating early in the poor group are precisely those which distinguish between Normal and logistic models in this group. It may be supposed that the poor suffer more disease than the not-poor and it is possible that some of the reported early menarches actually

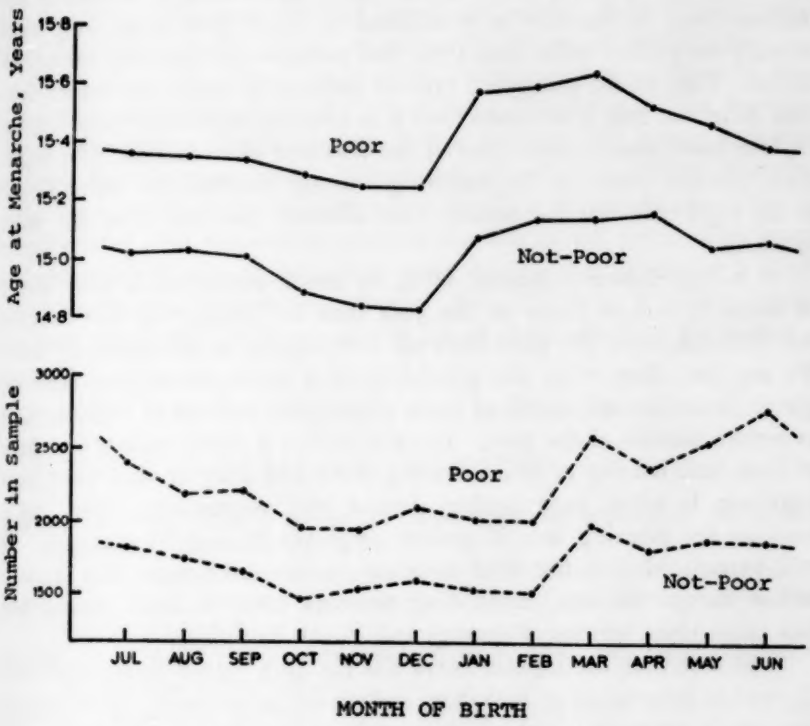

FIG. 5. MEAN AGE AT MENARCHE BY MONTH OF BIRTH.

represent bleeding or discharge from pathological causes rather than true menstruation. We have no positive evidence on this, however.

One major difference between the poor and not-poor groups lies in their habitual intake of animal protein. The not-poor girls benefit by receiving plenty of *amasi* (fermented milk) daily and a weekly ration of beef and poultry. In the poor kraals, with few cattle and fowls and little milk with which to make *amasi*, almost the only meat eaten is that from cattle which have died by accident or from disease.

The large apparent seasonal effect is hard to explain. Other published information on the topic is scanty, indeed non-existent for tropical or sub-tropical climates. We have examined the data of the 1959 survey of London schoolchildren (Scott, 1961) and find no sign of the phenomenon. Two conceivable explanations may be outlined. It might be thought that children born near the turn of the year were prone to errors of exactly one year in their stated ages, there being a tendency for children born in January to be allotted to (say) 1940 when they were actually born a few weeks into 1941, and perhaps vice versa for December births. This would produce a type of pattern of mean age rather like that which we find in our data; but it is unlikely that errors of this type explain more than a small part of the observed phenomenon. To begin with, random errors in the stated ages would tend to give lower slopes to the logit lines for the months most affected, and this does not seem to occur.

A straightforward seasonal effect, by which menarche is more likely to occur at certain times of the year than at others, will not explain our findings, since the girls were all interrogated at one point of time. We are left, then, with the possibility of a direct connection between age at menarche and month of birth, presumably because of malnutrition at certain seasons of the year. There is in fact a fairly regular shortage of food, and notably of *amasi*, during June and July of each year, and nutrition is often poor during August and September, when grain supplies are running low, if garden crops are limited by drought. It will be noticed that the total numbers in the sub-groups also show a rather abrupt increase, amounting to some 30% in each group, but this takes place between February and March birthdays.

The fact that the logistic curve fits the data rather better than the Normal is interesting on biological and statistical grounds. The logistic has certain properties, such as the possession of sufficient statistics, which give it some claim to be the simplest type of sigmoid to handle statistically. It is also simple to interpret, since the logit of a proportion p is merely the natural logarithm of the corresponding odds, $p/(1-p)$. Tanner, Whitehouse and Healy (1961) have found that the logistic gives a slightly better fit to data on the proportions of children of given ages who have passed successive stages of development in the bones of the wrist and hand. Hogben et al. (1948) also preferred the logistic to the Normal sigmoid for developmental data including age at menarche but on grounds that seem to us rather inadequate. The 1959 London menarche data are equally well fitted by either model.

SUMMARY

The distribution of age at menarche has been studied in 47,420 Bantu schoolgirls living in the Transkei Reserve, South Africa. The girls were interviewed by women Bantu teachers and asked whether or not menstruation had occurred. Dates of birth were taken from the school admission registers and are believed to be nearly all correct to the nearest month. The girls were divided into those coming from "poor" and those from "not-poor" homes, as judged by the school principals.

The proportions menstruating plotted against age were fitted by two alternative sigmoid curves, the cumulative Normal and the logistic. The latter gave a slightly better fit for the not-poor group and was superior for the poor group. There was an effect of month of birth, girls born early in the calendar year tending to menstruate some 4 months later than those born towards the end of the year. The mean age at menarche was 15.42 ± 0.04 years for the poor group and 15.02 ± 0.03 years for the not-poor group. In addition to the later mean, the poor group were more variable in age at menarche than the not-poor group.

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BOOK REVIEWS

A Drepanocitemia e a Antropologia. By J. H. SANTOS DAVID. 102 pp. Publicações Culturais No. 49. Companhia de Diamantes de Angola. Serviço de Saúde-Subsídios Para o Estudo da Antropologia na Lunda. Lisboa, 1960. Price not indicated.

This monograph reports data on the frequency of the sickle cell trait in the Lunda and Songo districts of Northeastern Angola. It also includes a rather comprehensive review and discussion of the work on the sickle cell gene, which is somewhat out-dated since the latest reference is 1956. The data on the frequency of the sickle cell trait are reported both by sub-district (concelho or circunscrição) and by tribe, and several attempts are made to determine whether or not the frequency of the sickle cell trait is associated with other factors such as sex, age, ABO blood groups, or malaria. The tribes appear to be closer to the actual breeding isolates than the districts, and there is great variability in the frequency of the sickle cell trait among the tribes, which range from 45.9% positives of 135 tested among the Bangala to 10.6% of 94 among the Luena with the other tribes (Lunda, Quiôca, Songo, Minungo, Xinge, Luímbe, Caconga, and Mataba) averaging around 20% positives. The frequency of 45.9% among the Bangala is one of the highest to be reported in any population.

The author is well aware of the problems raised by the high frequencies of the sickle cell gene in these populations, but the attempts to elucidate the factors involved are quite feeble. The author's conclusion that there is no relation between sickling and malaria is based on the absence of a significant correlation between the frequency of sickling and the incidence of malaria in the various sub-districts of the two districts, Lunda and Songo. But the incidence of malaria is measured by the number of hospitalizations, which are no measure of the endemicity of malaria, or by the spleen rate and parasite rate as recorded by sub-district, which are notoriously inaccurate. The precision of this work is comparable to others which have purported to disprove the sickling-malaria relationship, and this raises the point that any assertion of evidence against this relationship should be based on data that is at least as precise as the studies which have demonstrated it. Thus, the parasite rates and sickling rates should be recorded by realistic population units and not by districts or other administrative units which have little rela-

tion to the epidemiology of malaria or the breeding behavior of the human population.

Although the sub-districts are not realistic units, there is one significant correlation which seems to indicate the relationship of sickling to the operation of natural selection. I have found elsewhere in Africa that, other things being equal, there is some association between the frequency of the sickle cell trait and population density. This appears to be expectable in terms of population ecology theory since these populations would be closer to the upper limit of numbers which the environment can support, so that density-dependent factors should increase their toll of life. Thus, the correlation between the frequency of sickling and the population density among the sub-districts is significant ($r = .930$, $P < .01$) and seems to indicate differences in the amount of selection. The author's significant correlation between the frequency of sickling and the frequency of the blood group B gene may also be due to similar selection, but he interprets this correlation in terms of ethnic background. He then concludes that the variability in the sickle cell trait in this part of Angola is due to "racial heritage" and hence this gene has great "anthropological value."

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Readings on Race. STANLEY M. GARN. viii + 281 pp. Charles C. Thomas, Springfield, Illinois, 1960. $6.75.

This volume of readings records the belated impact of the New Systematics upon racial anthropology. Gone are the unproductive classifications of race, the construction of family trees, the tedious discussions of the standardization of techniques and measurements. Nowhere does the concept of morphological typing rear its specious head. Instead a series of papers published between 1952 and 1958 are reprinted to bring to anthropologists samples of the new trends of research current in the field of racial anthropology. It is of interest in terms of the breadth of the modern approach to race that more than one-half of the list were first published in journals that are not primarily anthropological in nature. The fact that biochemists, genticists, physiologists, and serologists are actively contributing to the problem is encouraging.

The selection of a series of readings to represent a broad and rapidly

changing subject is not easy. Preference is usually given to short, well-written papers that are not burdened by unnecessary technical complexity. In these terms Garn generally has chosen well. After a short introduction, the papers were organized into 9 sections, each of which is preceded by a brief commentary by the editor. His explanatory statements not only provide connections between the wide variety of included subjects, but give insight into Garn's own interests and into his attitudes toward racial anthropology. They could well have been further expanded.

The major topics revolve about the contemporary definition of race; evolution and race; race viewed from the point of view of climate, disease selection and genetic drift; natural selection and polymorphism; race mixture; and finally, experimental studies pertaining to racial differences. These topics are all important but do not comprise all of the forces operative in race formation. It is becoming more evident with each year that both cultural and ecological factors have their place in this area of inquiry. Garn is aware of this, as he has indicated in the preface. It is no fault of his that papers which are suitable for reprinting are rare. But the reader may well miss the important point that evolutionary factors in man are not limited to the biology of populations, but must and do involve cultural and environmental variables.

An editor has the implicit task of warning the reader about flaws, both technical and conceptual, which may be contained in his choice of papers. Thus Garn correctly points out in Section VII ("Genetic Drift and Race") that the early paper by Glass and his coworkers, on drift in a Dunker isolate, which is a classic in its own way, is marred by the fact that the West German population from which the group originated is not adequately known to demonstrate that genetic drift in fact had occurred.

A more serious criticism can be directed against Lasker's paper, "Human Evolution in Contemporary Communities." It is primarily concerned with estimations of rates of drift and gene flow, and these are made to seem easily obtained from normal census data. In fact, the proper values for substitution into the formulae are very difficult to derive, and this overy facile presentation errs in several serious technical ways. It is not a model for the proper approach to this complicated problem, and the editor should have so warned his readers.

But the reviewer's real quarrel is with Wilber's paper in Section V ("Climate and Race"). There is a tendency among those of us who work with natural populations of man to feel that experimental scientists, because of the control they customarily exert over the laboratory vari-

ables, are intellectually equally rigorous. Wilber sets up an unnecessary strawman and demolishes it with ill-used factual data and unacceptable evolutionary conceptualizations. This paper should not have been reprinted; better yet, it should not have been written.

In spite of the latter grumblings, Garn has edited a useful and timely series of readings. It may be hoped to have considerable impact in professional anthropological circles, especially among those whose knowledge of racial anthropology has not been upgraded from their own graduate student days. The editor has wisely described this volume as a case-book rather than a textbook; and it is. But it could well serve as a supplementary text in upper-division courses on modern racial anthropology. Garn should be encouraged to produce further editions, at five-year intervals, rather than waiting for a decade to elapse.

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Einführung in die Anthropologie. By HANS GRIMM. ix + 107 pp. Gustav Fischer Verlag, Jena, 1961. (Swiss francs 12.10 = ca. $3.00).

Professor Grimm is director of a new anthropological institute at the university of East-Berlin, had a medical and anthropological training and defines anthropology as the part of zoology concerned with man. His rather unusual and lively written "Introduction" is really a collection of 13 essays, forming only 92 pages of text. In the first 3 brief chapters he sketches the scope, aims and methods of modern physical anthropology by means of historical remarks and examples of results attained or hoped for. The remaining 10 chapters deal with the manifold usefulness of physical anthropology in allied sciences and for other interests, under the following headings (in as close a translation as possible and with reviewer's explanation): Role of anthropology in the foundation of social hygiene (anthropometric and genetic studies for the protection and correction of health); anthropology and sport (="Körperkultur"); anthropology as aid in legal problems (disputed paternity and identifications); role of anthropology in psychology (mostly constitution); anthropology as presupposition for sociology (chiefly parts of population biology); anthropology and art; anthropology as sister science of ethnography; anthropology as aid in pre-

history; anthropology as basic research in industry; and, finally, anthropology as an international science (= "völkerverbindende Wissenschaft"). There is also a welcome list of selected references of 8 pages.

Student will be deeply impressed by the wealth of facts, problems and theories, crowding these pages, but they seem to be expected to undrstand numerous unexplained technical terms. Experts will read most of these essays with interest and profit, but hardly without finding questionable statements. For instance, in the chapter on "the specializations of man from the viewpoint of comparative biology" it is claimed that man belongs to the systematic group of primates which is remarkable for its *special multiplicity of forms* and that within this group man is *distinguished by flattening of the trunk in cross-section, opposability of the thumb, reduced facial part of the skull, dentition without gaps,* besides of course bipedal locomotion, far-reaching loss of hair and a word-language. All the items, which have here been italicized, are certainly *not* distinguishing characters of primates or of man respectively.

English-speaking readers will be amused to find in the discussion of the bearing of the biology of human reproduction on sociological problems the ancient, joking definition of anthropology as "the study of man, embracing woman" solemnly translated as "Lehre von Mann und Weib und ihrer Umarung" (science of man and woman and their embracing) with the additional remark that Malinowski is supposed to have coined this very significant definition!

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Science and Medicine of Exercise and Sports. Edited by WARREN R. JOHNSON. xv + 740 pp. Harper, New York, 1960. $12.00.

Physical activity, like nutrition and aging, has a profound influence upon the human organism. It affects, in acute experiments, a multiplicity of functions and may leave chronic marks on body composition and body form. The appraisal of its effects on maintenance of health and longevity constitutes a continuing challenge as a research problem. In view of this broad spectrum of implications of physical activity for human biology, the appearance of a major, multidisciplinary treatise on the subject should not be left unnoticed. It supplements in important ways the currently available textbooks.

Among its forty-two contributors there are nationally and internationally known authorities in physiology, nutrition, physical education, psychology, and medicine. *In toto*, the volume is the best statement, in English, of our present knowledge of the various facets of this complex subject.

The book is divided into six sections. Since for the present purposes the psychological, cultural-historical and therapeutic aspects are less relevant, comment will be limited to the first three sections. Two of these deal with structural and physiological aspects of exercise and sports. In the third section, physical activity is considered in relation to man's life cycle, including maturity and aging.

In the part on body composition, various skinfold calipers are described. A regression equation (Pascal's) is presented for the determination of body density from three skinfold measurements and an equation (Keys and Brožek's) is given for the calculation of the total body fat from body density. A more detailed discussion of body density, its determination and its biological significance, would have been useful, as would be a presentation of the roentgenographic appraisal of body composition.

Some decrease in muscle mass in middle age and a sizeable increase in body fat were noted. These changes are due, at least in part, to the reduction of physical activity while the caloric intake remains the same. Several physiological functions relevant to performance capacity change as well. Maximal ventilation and maximal oxygen intake are both reduced when expressed per unit of body weight. For greater clarity it is desirable to express "fitness" of the cardio-respiratory system in middle-aged individuals in terms of maximal oxygen intake per unit of "active metabolic tissue."

In acute exercise of short duration, increase in hematocrit, blood hemoglobin and serum protein concentration can be observed. Vigorous running or cycling produce hemoconcentration and loss of plasma water to the interstitial fluid. As regards chronic physical activity, months in duration, hemoglobin and blood volume are higher in physically trained than in the untrained individuals. Habitual exercise, one in which an individual is engaged during the greater part of his life, is accompanied by lower values of body fat and, especially, by larger fat-free body mass.

Only limited information is provided on the anatomical and physiological differences between men and women, with emphasis on the psychological and cultural influences which determine the participation

of American womanhood in athletic activities. Further information is needed concerning the effects of the participation of women in athletic activities.

The limited data on exercise and its influence on human growth are well presented. Genetic and nutritional factors are considered. Androgen promotes growth of skeletal muscles whereas the female hormones inhibit the retention of nitrogen. This is reflected in the differences in strength between adolescent boys and girls.

Nutrition is one of the most important extrinsic factors affecting physical development. Improved nutrition accounts, in part at least, for the fact that the average American boy is 7% taller and 14% heavier than boys of the same age fifty years ago. Comparable data are known from Canada, England and New Zealand. Seasonal variations in growth are known, with spurt in height present in spring whereas autumn seems to be favorable to an increase in weight.

Hypertrophy of musculature, due to activity, is related to intensity of activity rather than to its duration. Chemical changes such as increase in glycogen, phosphocreatine, muscle hemoglobin and protein are induced by isometric rather than isotonic contractions.

Some evidence is at hand concerning the positive effect of activity on bone growth, mainly in animals. In man, heavy manual labor produces marked differences in anthropometric measurements (p. 458). Dominant hand or arm shows larger dimensions and also greater strength in comparison with the less used hand or arm. The mechanism through which activity influences growth is not know.

The present knowledge on longevity in relation to sports is meager. It seems that there is little difference in the life expectancy of college letter-winners and the non-athletic college graduates. But this is only one facet of a complex problem. Careful longitudinal studies comparing men who continue to be physically active with those who are relatively sedentary are badly needed.

Inactivity is the most important factor accounting for obesity in western societies. A table is included with caloric costs of different activities which could be used to balance out recreationally the caloric intake and the caloric output. Climate, effects of altitude, and aging are among the problems considered with reference to exercise.

The reviewer warmly recommends this volume to all who are concerned with the varied and profound effect of physical activity on human body in this era of immobilized human beings.

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Syllabus in Roentgenographic Cephalometry. By WILTON M. KROGMAN and VIKEN SASSOUNI. iii + 366 pp. College Offset, Philadelphia, 1957.

Clinical Cephalometry. By VIKEN SASSOUNI. iii + 248 pp. Philadelphia, 1959.

The Face in Five Dimensions. By VIKEN SASSOUNI. iii + 317 pp. Growth Center Publication, Philadelphia, 1960.

These three works are weighty accomplishments of the Philadelphia Growth Center, 1701½ Fitzwater Street, Philadelphia 46, Pennsylvania. Under Dr. Krogman's direction, this unit has contributed much to roentgenographic cephalometry. This technique involves making highly reproducible X-ray films of the human head in profile or full-face by means of a stabilizing frame or cephalostat. Skeletal or other outlines are traced from these films, and may be measured in a great many ways. Such dimensions often have about the same reliability from successive films as one finds in repeated measurements of the living head with calipers. For the past thirty years, orthodontists in particular have been fascinated with cephalometric tracings, and their clinical journals are glutted with such outlines of both harmonious and misshapen faces.

Programs for measuring these tracings are legion, and are described no better than in these three volumes by Sassouni and Krogman. This field is at about the point reached by craniometry in 1809, when von Török suggested that some 5,000 meassurements be taken on a skull.

Fortunately, Sassouni is not content to catalog the geometric conquests of his predecessors. Among his ingeneous accomplishments is a method for identifying unknown dead from eight measurements of posteroanterior cephalometric films.

In *The Face in Five Dimensions*, Sassouni elegantly displays shadow-contoured photographs of individual faces, along with cephalometric tracings of the same subjects. He has a rather original presentation of facial anatomy, although his descriptions are awkward in grammar, spelling, and the use of neologisms.

He describes the dispersion of facial bones and muscles around an imaginary center of the cranial base, and uses the awkward neologism "archial" or "archeal" to label this situation. It is not obvious to the reader whether this adjective refers to remote antiquity or to the shape of an arch. To Sassouni, the face resembles the Eiffel Tower, with horizontal portions corresponding to the occlusal plane, floor of the nose, and cribriform plate. The uprights of the tower less convincingly are compared with various vertical dispositions of facial structures. Sas-

souni's main intent here is to show that the face is built for maximal strength from a minimum of materials.

The Face in Five Dimensions includes some fascinating tracings of White, Negro and Chinese faces at various ages, facial types and their malocclusions, familial resemblances in facial form, and some serial tracings depicting the results of orthodontic treatment.

It is unfortunate that so much of the literature on cephalometric tracings takes up the spurious challenge of geometry rather than meaningfully attacking problems of morphogenesis. For example, orthodontists have perversely written more about the sella turcica than about the nose, despite the considerable relevance of the nasal cartilages to the origins of malocclusion. Cephalometric films might even be applied to studies of posture, occipital flattening, or the developmental relations of the inner ear.

In general, roentgenographic cephalometry has the same faults as other uncritical anthropometric rituals. Unless the measurer is seriously concerned with heredity, the etiology of cranial and facial deformities, polymorphisms, or evolution in general, his findings will fail to interest an informed and imaginative readership of human biologists.

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El Antigeno del Sistema Sanguineo Diego. By MIGUEL LAYRISSE and JOHANNES WILBERT. 160 pp. Editorial Sucre, Caracas, Venezuela. 1960. Paper. Price not indicated.

This is the first book to appear on the very interesting Diego blood group factor. As would be expected from authors who have been the leading workers with the new antigenic system, it is very competently written.

The book opens with brief discussions and definitions of terms such as antigen and antibody, and a discussion of genetics as it applies to blood groups. Discussions of the known blood group systems follow.

The treatment is primarily from the anthropological point of view, and the world distribution of each antigen is briefly discussed. The authors make use of the racial classification proposed by the present reviewer, and belong to the school, which they estimate to include about 60% of anthropologists, that believes in the anthropological usefulness of the blood groups.

The second part of the book is devoted to a detailed account of the discovery of the Diego system, its serology and genetics, and the distribution of the Diego gene in various populations. The final chapter of the book discusses the anthropological applications. The authors are commendably conservative in this section, and it is interesting to note that some of the observations that have appeared puzzling to those of us on the outside seem puzzling to them also.

There is a bibliography of more than 160 titles, and a brief appendix summarizing recent work. There is no index.

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*Lehrbuch der Anthropologia.** 3rd Ed. By R. MARTIN and K. SALLER. G. Fischer, Stuttgart, 1957.

I may be biassed, so I have to confess at the outset that to me Martin's *opus* never was a real "Lehrbuch," though very useful as a sourcebook in anthropology. This was true of the first edition (1914) (which I knew as a student) as well as of the second one (edited by Martin's widow S. Oppenheim, 1928). It is the task of this review to characterize the third edition in comparison with the previous ones, its improvements and deficiencies and flaws.

A short statistical survey reveals the following:

2nd edition (1180 pp. without the literature):		3rd edition (1478 pp. literature included):	
Introduction ("Allgemeines")	2%	General Anthropology	9.2%
Methods	7%	General anthropol. meth.	9.2%
Somatology:		Systematic Anthropol.	
1) Somatometric techniques and somatoscopy	10%	1) Methods	26.3%
2) Morphology of the body	29%	2) Nutrition	5.7%
3) Craniology and osteology	51%	3) Morphol. of the body	17.2%
		4) Osteol. (incl. craniology)	32.0%
		5) Central Nervous System	6.5%

* In this review the term Anthropology is used as a synonym for physical anthropology only.

The new edition contains some new chapters and it approaches a "Lehrbuch" a little bit more than the former editions. Nevertheless, it still clearly shows the intentions of its founder, the great systematic teacher and unifier of anthropological methods. The "Methods" comprise about 35%, and "Osteology" an additional 32%. Has modern anthology really only so little (33%) to teach about evolution, genetics, comparative anatomy of the Primates, age changes, gross tissue analysis, influence of cultural environment on evolution, races and human communities, and so on and so forth?

But let us consider some details. That the book begins with a short survey on genetics and some modern views on human biology is refreshing. However, one cannot agree with Saller's view on the definition of "Type" only from an etiological point of view. His classification of the suborder *Hominidae* in four genera seems obsolete, particularly the "genus Pithecanthropus" with three species. Saller's view on Fontéchevade is not clear (cf. pp. 77 and 90). But worse than some disputable viewpoints is Saller's non-recognition of the international rules of systematic nomenclature, which certainly does not befit a "Lehrbuch." Though Saller, one of the rare antinazi anthropologists in Hitler's "tausendjähriges Reich," has a sound and modern view on races, his presentation of racial systems is of historical interest only, revealing nothing new. The chapter on constitution is a very good one; in stressing the great practical importance of the constitutionological approach, he is quite right.

In the section on general methods one finds helpful advice for investigating the living as well as for treating skeletons, soft tissue, and employing photography, casts, fluorine and C^{14}. Reading about reconstructions of muscles one would expect Gerasimov's achievements to be mentioned at least; nevertheless, the most recent reference on this topic is from 1925!

The chapters on statistical methods seem to me very impressive and thorough, a great improvement as compared with the 2nd edition. There are even special chapters on genetical statistics but I do not feel competent to judge their quality.

Specific anthropological methods are dealt with as thoroughly as in 1st and 2nd edition. On pp. 277-8 80 instruments are enumerated, yet not a single caliper for skinthickness measuring among them! There are only 57 "most important" measurements. I do not agree with the mode of measuring the chest girth (which makes some sense in males only). Stadler's apparatus (from 1947) for measuring body volumes is declared as "the latest one" (p. 343)—where are the more

recent ones? I do not agree that the temperature of the water necessarily should be 25° Centigrade. Furthermore, density and specific gravity are not identical concepts or values, as it may seem on pp. 343 *et seq.* Measuring subcutaneous fat only right or left of the navel with a simple cephalometer, as indicated on p. 347, is a very unsatisfactory and outmoded technique. The statement that the subcutaneous fat thickness at the navel is the best one to judge the nutritional status is not correct (p. 348, *cf.* also pp. 416-7). Thus this particular section is behindhand, and was so even in 1957. The sections on capillar microscopy and finger nails are new.

The chapter on craniometric technique is as thorough as it was in the first two editions: on the skull 69 points of measurement are identified and a multiplicity of measurements and indices is described.

Among the physiological methods (some new ones) one finds some of importance for medical practice—Saller is an M. D., whereas Martin was not, and there are very practical nomograms. On pp. 643-649 there is a "Food Table" ("Nahrungsmitteltabelle") such as one would expect in a textbook on public health rather than in a textbook on physical anthropology. Finally, measurement blanks are added, including one for investigating cases of uncertain paternity.

The 2nd volume begins with an informative new chapter on nutrition, based almost solely on German literature. The section on Body build (255 p.) contains comparative data on Primates, age and sex differences, on ossification (without mentioning the Greulich and Pyle Atlas), on growth in height and weight and other characteristics, on retardation of growth in Hiroshima children, etc. There are, however, also some questionable and incomplete data. On specific gravity (pp. 871-2) Mies (1898) is quoted (though not cited in the literature) and Stadler (1947), but no American literature; no mention is made of the importance of such measurements for the problem of life expectancy. There are reproductions of the growth tables by Borovanský and Hněvkovský whose monograph is not cited in the literature (p. 993); on the other hand, the reviewer's monograph on the Development of secondary school students of Ljubljana is cited, together with two other papers (p. 1002), but no data are cited in the text.

The section on "Knochengerüst" is very large. One finds there very good ossification tables and every single bone is thoroughly described. There are facts a-plenty, but often without dynamic explanations.

Craniology was the hobby of anthropologists almost half a century ago. Later, anthropologists became aware of the fact that anthropology

could be a lively biological science; nevertheless, in Martin-Saller craniology still takes up 326 pp.—a fourth of the whole book. Unfortunately, in some tables (and on p. 1164 *et seq.*) it is not always clear whether the editor (Saller) is speaking about the head ("Kopf") or the skull ("Schädel"). In the section on "Craniology" one should not expect to find measurements on the living head, as is apparently the case in the tables on pp. 1169 and 1174. The comparative anatomy of the Primate skull is very useful. It is also more efficient to have the references appended to each bigger section instead of publishing them in a third volume, as was the case in the previous edition. The references in the text do not always agree with those in the "Literatur" (*e. g.*, Vlček, 1952 u. a. on p. 1215, and Vlček, 1953, on p. 1359). As for the skull index, there is still Struck's (1924) outmoded map. No information is provided on any Yugoslav nation in the tables on pp. 1236 to 1243 (except for one figure for living Serbs by Weisbach—from more than 60 years ago! The text to figs. 537a and b are reversed, the N. W. Queensland skull (537a) being that of an Australian and 537b that of an Eskimo!

The new chapter on the brain, comprises almost 100 pp. Particularly useful are the comparative figures on pp. 1496 to 1514; no less important is the discussion about the brain areas which are connected with various functions.

To sum up: Osteology comprises almost a third of the pages issued thus far. Apparently, it is the bones that are still the most interesting part of Anthropology. This partition happens to agree also with the deft definition of anthropology as "Stones, bones, and dirty stories about black people." We maintain that bones do not cover one third of this interesting science. Another third is dedicated to the "Methoden" (9.2%) and "Methodik" (26.3%). What is left? And what can be a layman's impression of what physical anthropology is about? Precisely as it was in the beginning of this century: An anthropologist is a queer stranger measuring skulls and bones with a caliper, his most precise and precious instrument.

I dissent. In my opinion, it would have been far better to leave old "Martin" as it was in 1928—a reliable sourcebook, and to translate into German, say A. Montagu's "Introduction" or E. A. Hooton's "Up from the Ape" or some other real *text*book. I think that even a revised edition of Saller's own "Leitfaden" (1930) would be a better "Lehrbuch" than the revised "Martin." A "Lehrbuch" should cover the basic facts, problems and methods of a science. Martin's "Lehrbuch der Anthropologie" (*the* textbook of anthropology as it used to be

called) has been a misnomer from the very beginning. It is an acceptable source book (a "Handbuch") on methods and it contains a lot of data which one cannot obtain elsewhere. But it is by no means a modern textbook of modern physical anthropology. I do not think it could be revised in this sense. It either is "Martin" or it is not. The "Martin-Saller" has lost some valuable characteristics of the first two editions, without gaining enough by Saller's co-authorship. I do not know any teacher who would be ready to say to his students "now read all these volumes of Martin (some 1500 pp.) and you will get a sense of contemporary physical anthropology." Most certainly they would not.

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Genetic Basis of Morphological Variation: An Evaluation and Application of the Twin Study Method. By Richard H. Osborne and Frances V. De George. Foreword by Th. Dobzhansky. xxii + 204 pp., 2 figures, 92 tables. Harvard University Press, for the Commonwealth Fund, Cambridge, 1959. $6.00.

This is an interesting and in some ways unique contribution to the study of the old problem of interaction between heredity and environment. It is based on 63 anthropometric measurements and indices, somatotyping, and masculinity-femininity ratings of 59 monozygous and 53 dizygous adult twin pairs. Analysis of variance is utilized throughout to evaluate intrapair twin differences, measurement error, interpair and sex differences. The results for each trait are given in terms of the proportion of the variance ascribable to the genotype and to the environment.

The emphasis of the book is on method, reflecting the need to discard the concept of a qualitative-quantitative distinction in genetic traits. Many traits once thought to be absolutely qualitative have been shown under critical analysis to vary quantitatively in strength of reaction. Genetic variability in man is largely polygenic so that methods must be adopted for studying complex multifactorial inheritance. There are three chapters evaluating the twin study method; surprisingly enough, there is no *absolute* method of determining twin zygosity. Four subsequent

chapters describe the design of the study. The analysis of normal morphological variation (actually less than half of the book) is contained in the remaining chapters on (1) stature, weight, ponderal index, (2) other body dimensions, (3) fat, bone, and muscle, (4) body build, and (5) masculinity and femininity. Following a short summary there are four methodological appendices.

Osborne and DeGeorge conclude that anthropometric measurements differ widely in their value for genetic studies. In general there is a relatively greater genetic component of variability in postcranial measurements taken parallel with long axis of the body. Measurements of bone (wrist breadth), fat (thigh and scapular skinfolds), and muscle (upper arm diameter) are considered to provide satisfactory indications of a genetic component of variability. Genetic and environmental components of variability are shown in stature, but unlike previous studies on children, no genetic component is found in weight. There is mention of a possible hereditary component in some undefined "gross size factor," but this factor confuses the components of body composition. It would be well to test relative body weight, lean body mass, or fat-free body. In Sheldon's somatotype, "the genetic aspect . . . rests primarily upon the ectomorphic component." While head length does not show a genetic component of variability, the cephalic index "provides the largest MZ:DZ [monozygous: dizygous] ratio of all indices calculated" indicating an exceedingly strong genetic component.

Some measurements provide significant measures of genetic variability in males but not in females, and vice versa. Although this may be expected in certain instances, in most cases it makes no biological sense. This phenomenon seems to reside in the statistical analysis. Male and female data have been analyzed separately, *including measurement errors.* The authors have not pooled male and female measurement errors (ME) where it is legitimate to do so. In at least ten separate measurements male and female ME variances shown no significant difference (at .025 significance level used by the authors as criterion.) When the male and female ME variances are pooled, environmental interaction is shown statistically for ten measurement sites (upper arm length, middle finger length, forearm circumference, sitting height, suprasternal height, biacromial breadth, chest breadth, bi-iliac breadth, chest circumference, and total leg length) *in both sexes,* although Osborne and DeGeorge found environmental interaction only in the female (See table 1). In the masculinity-femininity ratings, environmental interaction, previously demonstrated only in the male is now shown in both sexes. These obser-

TABLE 1

Reanalysis of Environmentally Influenced Variability

(Data from Osborne and DeGeorge)

TABLE #	MEASUREMENT—INFLUENCE SHOWN IN ONE SEX ONLY	F	s^2	ME : MZ F RATIO	P	ENVIRONMENT INTERACTION Osborne DeGeorge	ENVIRONMENT INTERACTION Reanalysis pooled ME
15	Upper arm length	1.02	.111	♂2.85	.005	female	both
				♀3.32	.005		
18	Middle finger length	1.33	.008	♂2.63	.01	female	both
				♀2.88	.005		
22	Forearm circumference	2.44	.097	♂7.34	.001	female	both
				♀3.57	.001		
26	Sitting height	2.19	.110	♂4.75	.001	female	both
				♀4.00	.001		
27	Supraasternal height	2.15	.251	♂3.47	.001	female	both
				♀3.69	.001		
29	Bi-acromial breadth	1.46	.256	♂2.81	.01	female	both
				♀3.61	.001		
30	Chest breadth	1.44	.109	♂3.95	.001	female	both
				♀5.67	.001		
34	Bi-iliac breadth	2.29	.102	♂5.21	.001	female	both
				♀5.81	.001		
37	Chest circumference	2.15	.955	♂5.58	.001	female	both
				♀6.16	.001		
42	Total leg length	2.00	.154	♂5.72	.001	female	both
				♀5.82	.001		
67	Nose breadth	3.00	.010	♂1.80	.10	male	none
				♀1.50	.25		
90	Masculinity-femininity rating	1.45	9.077	♂2.99	.005	male	both
				♀2.04	.025		
17	Hand length	1.45	.081	♂1.73	.10	female	female
				♀4.11	.001		
66	Interocular width	2.67	.007	♂3.57	.001	male	male
				♀1.57	.25		
23	Minimal wrist circumference	7.75	—			male	
44	Lower leg length	4.31	—			female	
58	Total face height	5.75	—			female	
63	Minimal frontal breadth	3.14	—			female	
64	Bizygomatic breadth	9.00	—			male	
69	Mouth width	3.57	—			female	

F = test of equality of male and female measurement error (ME) variances, where F (.025) = 3.13

s^2 = pooled male and female ME variance

P = probability level of ME: MZ F ratio, where $P \leqq .025$ is criterion of environmental interaction

vations, largely a reflection of small sample sizes (e.g., 7 for the male ME variance) call for substantial changes in the author's conclusions.

This book contains none of the original data, not even means or medians. This is a major limitation, for the inquiring reader is not able to check the statistical manipulations against the primary data. The translation of statistical inference into biological meaningful significance is a task not fully accomplished by Osborne and DeGeorge. Why is it that while head length "is of limited value in indicating genetic variability," when it is combined with head breadth, the cephalic index "provides a highly significant test of genetic variability?" Conversely, muscle, bone, and fat measurements independently indicate genetic variability, yet the mesomorphic and endomorphic somatotypes show no significant genetic variability!

Measurement of the genetic basis of morphological variation is indeed difficult in man. Osborne and DeGeorge have made a step in this direction. But the limitation the authors noted in previous twin studies, namely the "preoccupation with the problem of establishing the relative importance of heredity or environment rather than that of understanding their interactions . . ." is still far from overcome.

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Natural Selection in Human Populations. Edited by D. F. ROBERTS and G. A. HARRISON. viii + 79 pp. Pergamon Press, London, 1959. $3.00.

Six papers, contributed by L. S. Penrose, A. R. G. Owen, C. A. Clarke, R. M. Sheppard, T. Dobzhansky, and E. H. Ashton, were presented at the second (1958) Symposium for the Study of Human Biology. Two authors (Dobzhansky and Ashton), although they describe studies in non-human populations, nevertheless point out the kinds of difficulties which have been encountered and the experimental approaches which have proved fruitful. The discussion of coadapted gene systems in Drosophila and its implications for human populations is particularly interesting. The other four papers deal specifically with aspects of selection in human populations.

Actually, only three specific topics directly concerned with man are

analyzed in any detail. Mathematical models, which describe the effect of selection on multiple allelic and multiple locus systems, are presented. It is apparent, as the author points out, that efforts in this direction have just commenced, and much more is required if any comprehensive mathematics of this important area is to be developed. There is a good discussion of the estimation of fitness of various genotypes which is of obvious interest both in this connection and in the analysis of mutation rates in man by the indirect method. A careful presentation of the rather intricate relationships of blood groups and disease points out the need for further study both in this system and in the interaction of other ostensibly neutral genotypes and disease. Penrose, in his general review, isolates certain problems which may yields coherent answers, and also discusses aspects of the genetics of polymorphism.

Penrose's documentation of areas needing exploration, engendered a sense of frustration which continued throughout the book. This was due to the clear evidence that abysmally little is actually known about the *mechanisms* of natural selection. The direction of further investigation is made amply clear. Fine examples of polymorphisms, apparently maintained by selection pressures, abound in man and in experimental organisms. The inability, however, to come to grips with the specific agencies which initiate the polymorphisms and maintain them at particular frequencies is all too evident.

The contents of this book will be valuable to all biologists, and especially to human biologists, in terms both of data and of concepts. Experiencing the associated frustration also has merit! It is apparent that the remarkable recent advances in molecular genetics must also be interpreted in terms of formal evolutionary mechanisms. These, in turn, evidently require interpretation in terms of the action of highly specific selective agents. We must hope, with the contributors to this Symposium, to get beyond the strictly formal level, where so frequently and blithely unexpected phenotypic distributions are attributed to "natural selection."

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Quantitative Zoology. Revised edition. By GEORGE GAYLORD SIMPSON, ANNE ROE, and RICHARD C. LEWONTIN. vii + 400 pp., Harcourt, Brace and Company, New York, 1960. $8.00.

In 1939, George Gaylord Simpson and Anne Roe produced the first edition of *Quantitative Zoology.* At that time it was one of a series of works, including *The New Systematics* (edited by Julian S. Huxley, Oxford, Clarendon Press, 1940), which reflected a fundamental change taking place in biological sciences.

Quantitative Zoology represented the very forefront of basic biological thinking in 1939. There are many indications that the revised edition, though cognizant of some of the changes which have occurred in the intervening 20 years, has not kept pace with the methodological developments in quantitative biology in spite of the addition of a third author who has specialized in biometrics as well as zoology.

Since the book deals with situations in zoology where quantification is appropriate, it is worth pointing out that the variety of measures made possible by modern instrumentation could be amplified. For instance, color measurement need not be restricted to tint photometers or color tops. Because of the availability of easily usable and relatively inexpensive photoelectric measuring devices, the claim can no longer be made that ". . . this is such an elaborate and exacting process that it is impractical in most zoological work" (p. 27). Hematological characteristics and amino acid-creatinine proportions (as excreted in urine) are also omitted from consideration although their measurement is not difficult by modern methods. This slighting of measurements related to the more biochemical aspects of organisms is understandable in view of the predominantly palaeontological backgrounds of the authors, but when the close relationship of such aspects with basic genetic material is recognized, they should be mentioned in any modern quantitative approach to biology.

The really solid and enduring contribution made by this book is in the area of the logic and application of statistics in reference to zoological problems. The approach and format utilized in 1939 are preserved with a few changes. The addition of a chapter dealing with the analysis of variance written with the same thorough understanding that characterized the other sections is the most obvious change.

There are two less evident but more pervasive improvements which influence the whole presentation. The basic logic of each quantitative procedure is sharpened and the presentation further clarified. Sample

size is no longer assumed to be infinite except in special cases. Furthermore, each technique is presented with the assumption that the potential user will have access to a desk calculator. Thus brought to to date, there is no other published source which can give professional and beginner alike so clear and well-reasoned a picture of basic statistical procedures and their applications.

Chapter 8, originally entitled "Probability and Reliability," dealt only very cursorily with the problems of the relative frequency with which specific events occur, given an infinity of trials. In the revised edition, this chapter has a discussion of probability and various kinds of distributions. Considering the amount of quantitative sophistication necessary to properly understand the logic of "Probability and Probability Distributions," as Chapter 8 is now entitled, it is a considerable improvement to find the discussion of *binomial, Poisson,* and *normal* distributions deferred from its original position in Chapter 4, where it was uneasily included with a consideration of the mechanics of data grouping.

However, the assumption is made that most zoological data are normally distributed and the quantitative procedures discussed are those designed to handle such data. Although the existence of techniques for use with non-normal distributions is acknowledged, the reader is left with the impression that these are forbiddingly difficult and useful only to "professionals." Reference is made in the text to "Mood (1950, Chapter 16)" (p. 158) as a source on non-parametric statistics, but this is included in neither bibliography nor index. The complete reference should have been, Alexander McFarlane Mood: *Introduction to the Theory of Statistics.* McGraw-Hill: New York, 1950.

It would have taken only a very brief space to have mentioned the basic assumptions and utility of a non-parametric approach. As for the assertion that such a technique is "quite weak in the precision with which inferences can be made" (p. 158), the very author to whom they incompletely refer has shown that the Mann-Whitney U test, an exceedingly powerful and usable technique, is close to being 95% as efficient as the t test, and of course, not tied to the distribution assumptions made by the latter.

Unfortunately, the expanded concern with the probability of the occurrence of events is too brief and does not adequately distinguish between the probability of the successive occurrences of single events and the probability of the simultaneous occurrences of more than one event. The logic behind the outcome of coin-tossing where one coin is involved is indicated, but the same passage also refers to the tossing of more than

one coin, yet the logic of the probabilities involved is not explained. This omission may seem unimportant in a book that otherwise shows a complete understanding of the principles and problems of quantification, but it foreshadows further omission which should be of concern to all who maintain an interest in mid-20th century biology from a quantitative viewpoint.

While the enew edition considers analysis of variance, no mention is made of such complex but important techniques as Fisher's discriminant function, Rao's D^2 and some form of factor analysis. Admittedly a full discussion of such matters with directions for their use would have rendered the book undesirably long, yet some discussion of the basic theory behind and utility of several forms of multivariate analysis along with appropriate references to more detailed sources would have greatly enhanced the value of the volume.

Finally, although the general use of desk calculators is assumed, no mention is made of modern data processing systems. Punch cards and tape methods of data handling, high speed computers, service bureaus and libraries of machine programs have been effecting a revolution in quantitative procedures. Methods of scaling aimed at reducing raw data to forms readily fed into computers are overlooked. The fact that machines can compute all kinds of regressions, correlation problems, and variance analyses in a fraction of the time and expense necessary to do them by hand is not mentioned.

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A NEW GRAPHIC METHOD FOR THE EVALUATION OF THE TEMPO AND HARMONY OF PHYSICAL GROWTH OF CHILDREN:

The Method of Developmental Channels and Steps

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AMONG the many methods of evaluation of growth of children (Correnti, 1949; Deriviere, 1953; De Toni, 1954; Lelong, 1956; Baszkirow, 1956; Kapalin, Prokopec and Prošek, 1957; Perkal, 1958; and Wolański, 1961a), the only one which permits an examination of the rate of development is that of Wetzel (1947). Unfortunately Wetzel's method is based on too many *a priori* assumptions. Those who criticize it cite that: it is based on the overly formalistic allometric principle, it uses the same grid for both sexes, the pattern of channels is purely hypothetical, the details of the American material upon which it is based are unknown (hence the population of reference is uncertain), and the system of horizons does not correspond with the distribution of the ratio of height to weight nor take into account differences in maturation among different somatic types (Correnti, 1949; Cotellessa and Salomone, 1951; Perkal, 1958; Wolański, 1959). The present author therefore undertook to present his method of analyzing the rate and harmony of growth. The method makes possible the examination of the rhythm of development, the differences in rate of development in successive periods of ontogenesis. Different types of development are characteristic of different bodily types.

A grid has been worked out on the basis of the method presented in this paper. It uses data (Tables 1 and 2), collected by scientific workers of the Mother and Child Institute of Warsaw, on healthy skillful children in Warsaw, brought up under good conditions. Martin's techniques and equipment (Martin-Saller, 1957) were used for children over two years of age. For length measurements of infants, the method and instrument (called *liberometer*) devised by Wolański was used. Statistical calculations were done by Guilford's (1956) formulas.

TABLE 1

Body height, body weight and chest diameter (at the level of xiphoidale) of Warsaw boys in 1959

AGE IN YEARS	BODY HEIGHT		BODY WEIGHT		CHEST DIAMETER		N
	$\bar{x}$	σ	$\bar{x}$	σ	$\bar{x}$	σ	
0	51.11	2.35	3.4183	0.526	—	—	164
3	96.35	4.26	15.11	1.65	52.25	2.18	111
4	101.27	4.60	16.54	1.88	53.53	2.12	94
5	106.71	5.10	18.01	2.12	54.99	2.65	105
6	113.35	5.58	20.35	2.63	56.71	2.64	111
7	121.39	5.26	23.72	2.77	58.50	2.87	127
8	125.79	5.52	25.49	3.09	59.26	3.02	110
9	130.71	5.66	28.28	4.63	61.36	3.22	116
10	135.75	6.76	31.38	5.94	63.42	4.17	113
11	139.91	5.60	34.47	6.10	65.26	5.25	105
12	146.17	7.52	38.37	6.71	67.68	4.55	100
13	151.99	8.68	42.94	8.03	70.21	5.39	101
14	158.75	9.48	48.75	8.13	74.05	6.22	100
15	166.59	6.88	57.29	9.64	78.74	6.76	102
16	170.41	6.48	60.17	7.20	81.76	5.02	100
17	171.89	5.94	62.07	6.42	83.99	5.08	101
18.5	172.99	4.88	64.05	6.59	85.38	4.51	61

TABLE 2

Body height, body weight and chest diameter (at the level of xiphoidale) of Warsaw girls in 1959

AGE IN YEARS	BODY HEIGHT		BODY WEIGHT		CHEST DIAMETER		N
	$\bar{x}$	σ	$\bar{x}$	σ	$\bar{x}$	σ	
0	50.40	2.47	3.3387	0.508	—	—	160
3	96.03	4.46	14.88	1.61	51.76	2.29	113
4	99.33	4.44	15.65	1.91	52.06	2.53	106
5	107.21	4.38	17.91	2.13	53.99	2.32	103
6	112.71	4.40	20.04	2.51	55.38	2.33	106
7	120.61	5.24	23.34	3.87	57.01	3.49	136
8	123.95	5.48	24.68	3.82	58.00	3.60	108
9	130.45	5.98	28.54	4.92	60.11	3.88	123
10	133.81	6.42	31.21	4.08	61.60	4.67	103
11	140.73	7.28	35.62	6.68	64.20	5.16	135
12	148.55	6.48	43.07	9.10	67.74	5.72	118
13	152.05	6.64	43.21	8.05	68.59	5.11	110
14	156.53	5.96	49.28	7.52	73.01	5.71	104
15	159.63	5.78	53.32	7.08	74.18	4.70	102
16	159.81	5.72	55.10	8.21	75.74	5.74	109

ASSUMPTIONS AND PRINCIPLES OF THE METHOD OF CHANNELS AND STEPS OF DEVELOPMENT

The following assumptions underlie the method of channels and steps of development.

Height and weight are not only representative of the growth process, but also, to some extent, of the complex phenomena of physical development. The interrelations of changes in height and weight can serve as an index of differentiation (Garn, 1952).

The course of development should be considered as good when the developmental curve of an individual, as plotted on the grid described below, runs parallel to the borders of its channel; it should be considered poor when it wanders between channels. This assumption is based on the belief that periodically alternating " springing-up " and " filling-out " are not healthy phenomena and that normal increases in height and weight are harmonious (Jasicki, 1938, 1948a, b; Bocheńska, 1958).

Average values may be taken as optimal when they are taken as the arithmetic mean of normally distributed values in a population with desirable endogenous factors—children born at full term of healthy parents Stołyhwo, 1955)—and exogenous factors—such as home conditions and hygienic standards.

When the distribution is normal, we accept $\pm 0.5\sigma$, which encompasses 38% of individuals, as the arbitrary limits of the optimal values.

Since there is a high degree of correlation between growth in height and weight (Aron, 1940; Jasicki, 1948a, b), these two measurements serve well to divide children into three categories: slender, harmonious, and stout.

Using deviation units of σ_h, σ_w (Guilford, 1956) at each age, one can connect points to define limits for a series of channels MD, MC, MB, MA, M, Ma, Mb, Mc, Md for males, and KD, · · ·, Kd, for females, in which σ_h, σ_w is the standard deviation of height (h) and weight (w) and A, B, C, and D represent the progressively slenderer and a, b, c, and d the progressively stouter channels (figures 1 and 3).

Standard levels of development were then determined by dividing the change in the values of height and weight from birth to 18.5 years in boys, and from birth to 16 years in girls, into 100 equal sections (marked by intermittent lines on the grid). The number of standard levels comprised within each year's actual average growth can be seen by reference to the uninterrupted oblique lines in the grids. The number of standard levels an individual child passes through in a given period

of time (NSLI) can be multiplied by 100 and divided by the average number of levels for children of his position (NSLA) on the grid (figures 1 and 2). This is called the index of developmental rhythm

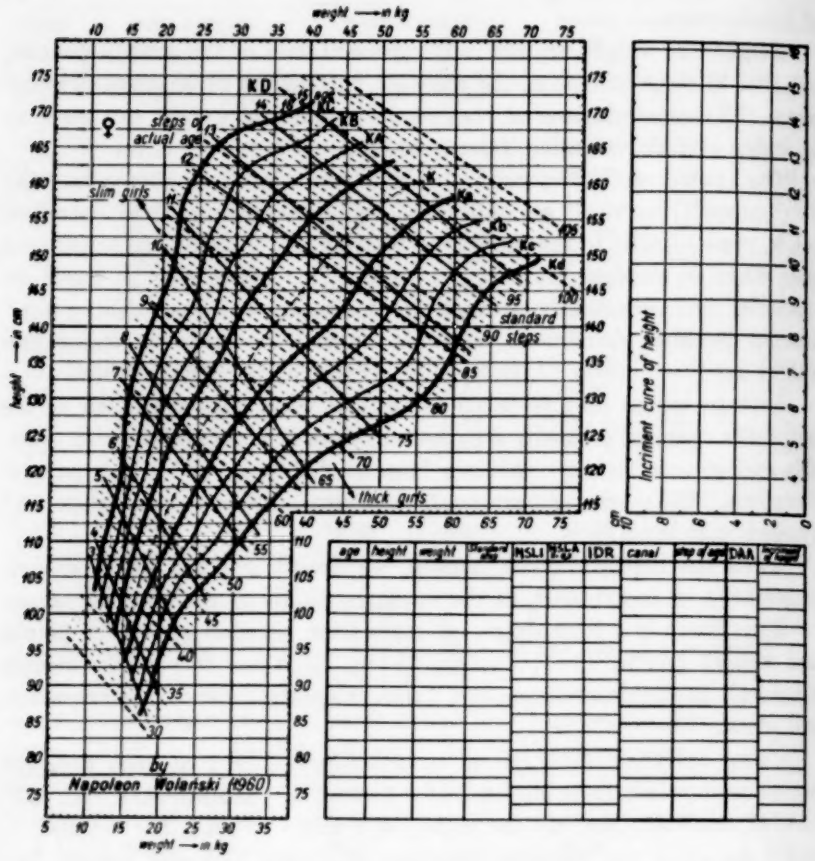
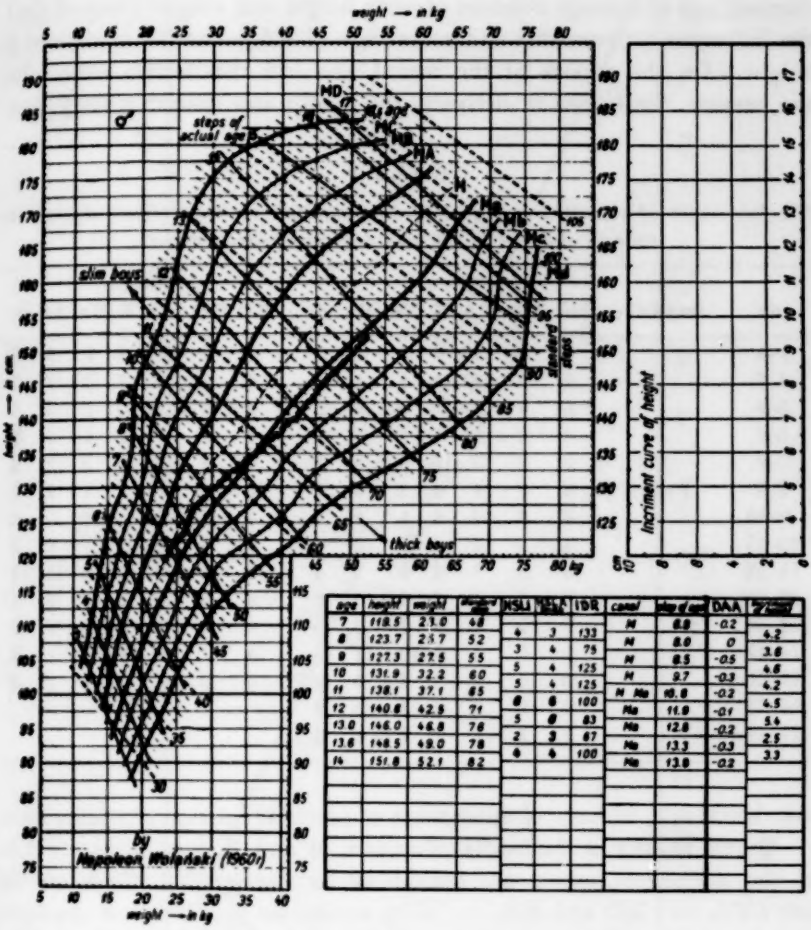


FIG. 1. THE GRAPHIC METHOD OF EXAMINATION OF THE TEMPO AND HARMONY OF GROWTH—GIRLS.

(IDR). Since the average number of standard levels achieved each year differs, depending on the channel, it is clear that the interrupted lines representing standard, and uninterrupted lines representing average achieved growth will not be parallel to each other. The higher the index of developmental rhythm, the faster the rate of growth relative to other children. If a child is below the average chronological level, but the

index of developmental rhythm exceeds 100, the child is compensating for his retardation.

Insofar as the dynamics of growth can be interpreted from changes



age	height	weight		NSLI		IDR	canal	step of age	DAA	
7	118.5	21.0	48				M	8.8	-0.2	
8	123.7	25.7	52	4	3	133	M	8.0	0	4.2
9	127.3	27.5	55	3	4	75	M	8.5	-0.5	3.8
10	131.9	32.2	60	5	4	125	M	9.7	-0.3	4.8
11	138.1	37.1	65	5	4	125	M Ma	10.8	-0.2	4.2
12	140.8	42.5	71	8	8	100	Ma	11.9	-0.1	4.5
13.0	146.0	46.8	76	5	6	83	Ma	12.8	-0.2	5.4
13.6	148.5	49.0	78	2	3	67	Ma	13.3	-0.3	2.5
14	151.8	52.1	82	4	4	100	Ma	13.9	-0.2	3.3

FIG. 2. THE GRAPHIC METHOD OF EXAMINATION OF THE TEMPO AND HARMONY OF GROWTH—BOYS.

in height and weight, the grids for boys (Fig. 1) and girls (Fig. 2) permit the evaluation of both individuals and populations. A point representing the height and weight of the child is marked on the grid each time the child is measured and when these points are joined one

gets a curve representing the development of the individual. Besides the graphic changes, there is room on the face of the grid for a table of age, height, and weight of the child, achieved "Standard step," rate of standard steps (NSLI), Index of developmental rhythm (IDR), channel, age of average children of same height and weight (step of age), the difference between this and actual age (DAA), and the increment in height. On the reverse of the record, one can insert data concerning the parents, the course of delivery, the health and disease history, bone

TABLE 3

The increment of standard levels (NSLA) occurring on the average during one year in children of various degrees of disharmony.

		BOYS							GIRLS						
AGE	CHANNEL:	MC	MB	MA	M	Ma	Mb	Mc	KC	KB	KA	K	Ka	Kb	Kc
3–4		4	4	4	4	3	3	3	2	2	2	3	3	3	3
4–5		5	5	5	4	4	4	4	6	6	7	7	7	7	7
5–6		5	5	5	5	5	5	5	5	5	5	5	5	5	5
6–7		6	6	6	6	7	7	7	7	7	7	7	7	7	7
7–8		3	3	3	3	3	3	3	4	4	4	3	3	2	2
8–9		3	3	4	4	4	5	5	6	6	7	7	7	7	7
9–10		4	4	4	4	4	4	4	6	5	5	4	3	3	2
10–11		2	2	3	4	4	5	5	6	6	6	7	7	8	8
11–12		8	8	7	6	6	5	4	7	8	9	10	10	11	12
12–13		6	6	6	6	6	6	6	4	4	3	3	2	2	1
13–14		8	7	7	7	7	6	6	6	6	6	6	6	6	6
14–15		5	6	7	9	10	11	12	4	4	4	4	4	4	4
15–16		6	5	4	4	4	3	2	0	0	1	1	2	2	2
16–17		2	2	2	2	2	2	2							
17–18.5		1	1	1	2	2	2	3							

and dental age, and age of appearance of secondary sexual characteristics.

Curves falling in the medial channels, M and K, *i. e.*, within $\pm 0.5\sigma$ of the arithmetic mean, are considered as harmonious, those on the left (MA, · · · MD and KA, · · · KD) as slender and those on the right (Ma, · · · Md, and Ka, · · · Kd) as stout. Deviations as great as MD, KD, and Md, Kd should be considered as very disharmonious and suggest inquiry into the cause. If a child maintains the same channel, however, it suggests a constitutional slimness or stoutness whereas "wandering" from channel to channel suggests some irregularity from a consistent pattern of growth in height and weight (see Jasicki, 1938, 1948a, b and Bocheńska, 1958). Similarly, in respect to chronological level, a child

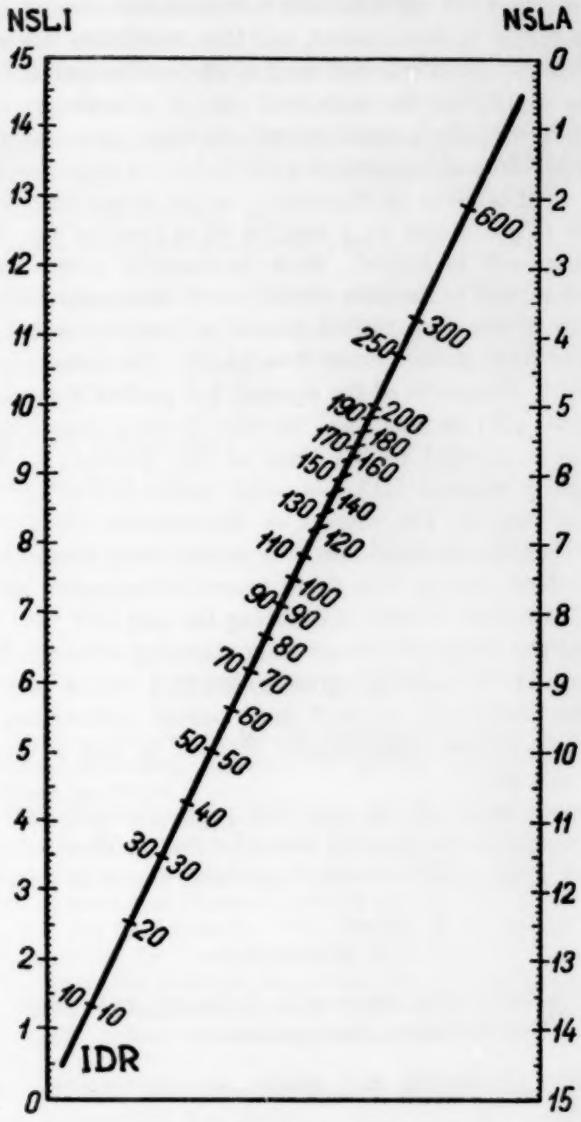


FIG. 3. CALCULATION OF THE INDEX OF DEVELOPMENTAL RHYTHM (IDR) FROM THE NUMBER OF STANDARD LEVELS PASSED THROUGH PER YEAR BY THE INDIVIDUAL (NSLI) AND THE AVERAGE OF SUCH LEVELS PER YEAR ACHIEVED BY CHILDREN OF HIS POSITION (NSLA).

of, *e. g.*, seven years of age who does not reach the step of age 7 in his channel is retarded in development, and this retardation can be expressed in years (DAA). Similarly the rhythm of development (IDR) can be evaluated by comparing the individual rate of achievement of standard levels (NSLI) with the general rate of achieving them during the same age span by children of the same sex (NSLA). A nomograph for interpolation of IDR is given in Figure 3. In the upper right-hand corner of the grids (Figs. 1 and 2) a small grid is inserted for plotting the incremental growth in height. Such incremental curves manifest the adolescent spurt and permit the evaluation of this maturational stage.

In Figure 2 data are plotted (small circles) for a boy, ZW, who was examined during the ages of 6 to 14. 1. His development at first runs parallel to the limits of the channel but gradually passes from the medial channel (M) to that (Ma) for slightly stout boys where it again turns and runs parallel to the limit of the channel; 2. The boy is initially slightly retarded (DAA = — 0.2 years) and at age 14 is again similarly retarded; 3. The rhythm of development (IDR) is initially more rapid than characteristic for this period, then, from 8 to 10 years of age, it is about average, it is again somewhat increased, lowered again from 11 to 13.6 years of age, and during the last half year is average; 4. In general the deviations do not seem alarming although the retardation at about 11–12 years of age was due to a rather serious disease. The progress should be watched until sexual maturation which, to judge from the increased increments between 13 and 14 years of age, appears to be near.

The present grids can be used for populations of similar anthropological composition to those of Poland. For children of other areas, however, new grids could be worked out using the same principles.

SUMMARY

New grids for evaluating growth in height and weight have been constructed. The following assumptions are made:

1. Changes in height and weight are representative of general development;

2. The developmental course is better when it parallels that of the average of healthy, skillful children;

3. The type and degree of disharmony can be measured in standard units (σ) of deviation in height-weight ratio;

4. The level of chronological developmental can be determined from the coefficient of correlation;

5. The division of the whole growth into 100 equal parts provides standard levels;

6. Growth rhythm can be evaluated by comparing standard levels achieved in an individual child with those achieved on the average by other children during the same period;

7. The sexes should be considered separately.

The author examined 3,500 Polish children of ages 3 to 18.5 and 324 new-borns and determined standard levels and constructed grids for boys and girls separately. The grids permit evaluation of child development with regards to:

1.) degree of disharmony;
2.) developmental level;
3.) growth conditions; and
4.) developmental dynamics with regard to rate and rhythm.

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THE CONCEPT OF RELATIVE LIMB DOMINANCE

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LIMB dominance has been defined as left or right "handedness" for any manipulation performed by the upper extremity. It encompasses a wide range of movement from the fine manipulation of the thumb to the sweeping movements of the shoulder. The gradations of ambihandedness between the two extreme positions of left and right dominanace have been difficult to define because they involve some manipulations with the opposite limb. It might be better to analyze the total pattern of movement necessary for positioning the limb. By observation, one finds that this movement is a combination of rotation and circumduction in a medial direction, occurring simultaneously at four separate joint levels: the shoulder, the elbow, the wrist and the thumb.

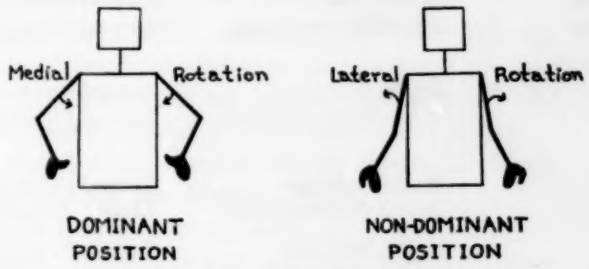

FIG. 1. BASIC LIMB DOMINANCE.

If one examines the range of rotation and circumduction movements for the extremity, one finds that two extreme positions are possible: that position following extreme lateral rotation and circumduction (a lateral rotary movement), which places the limb into the rather helpless human anatomical position; and the opposite position following extreme medial rotation and circumduction (a medial rotary movement) which resembles the positioning of the limbs for quadrupedal locomotion. The medial rotary movement represents the active dominant positioning; the lateral rotary movement represents the inactive non-dominant positioning. See figure 1.

If one were to apply the theory of absolute limb dominance, as expressed preferable by Humphry (1861), one might expect to see all involved joint levels, from the shoulder to the thumb, simultaneously rotated and circumducted medially to the limit of the range of movement. However, because of the freedom of movement at each of the several joints levels (Steindler, 1955), many mixed patterns of dominance are possible. In any unimanual act, one arm is maneuvered into the more dominant position, while the other arm remains in the relatively less dominant position. According to the theory of relative limb domi-

TABLE 1

Analysis of limb rotation

LEVELS	DOMINANT POSITION	NON-DOMINANT POSITION
Shoulder		
Clavicle and	Forward Rotation	Backward Rotation
Scapula	Forward Circumduction	Backward Circumduction
Humerus	Medial Rotation	Lateral Rotation
Humerus	Medial Circumduction	Lateral Circumduction
Elbow		
Radius	Medial Rotation	Lateral Rotation
Ulna	Medial Circumduction	Lateral Circumduction
Wrist		
Radius	Medial Rotation	Lateral Rotation
Carpus	Medial Circumduction	Lateral Circumduction
Thumb	Medial Rotation	Lateral Rotation
	Medial Circumduction	Lateral Circumduction

nance, the total pattern of movement for one extremity may not be so dominant nor the other so non-dominant as might be expected.

If one examines table 1 to compare the movements involved at the separate joint levels, one finds that all four joint levels participate in both rotation and in circumduction to varying degress. There are 360° of rotation possible: 90° at the shoulder level, 180° at the combined elbow and wrist levels and 90° at the thumb level. There is roughly a circular form of circumduction accompanying rotation at each joint level, except at the elbow level where the movement is elipsoidal. This elipsoidal circumduction accompanying the rotation is possible because of the much greater moment of flexion-extension (150°) than of adduc-

tion-abduction (8–9°) (Ray, Johnson, and Jameson, 1951). When, for some reason, there is limitation of the freedom of movement at one or more joint levels, there may be compensatory rotation and circumduction of the other joint levels (Steindler, 1955).

Dominance Level Tests:

The theory of relative limb dominance has been advanced by utilizing a combination of long objective questionnaires and performance tests (Rife, 1922; Downey, 1927; Woo, 1928; Ojemann, 1930; Koch, 1933; Leche, 1933; Durost, 1934; Newman, 1934); they indexed handedness according to the ratio of right to left responses. Hellbrandt and Houtz, 1950 differentiated handedness by the relative degree of fatigability. No one has yet systematically classified "handedness" according to patterns of joint level dominance.

All the limb dominance tests for this study were devised to compare limbs at each joint level during the initiation of simple simultaneous movements. By comparing the positions of the two limbs at the completion of the movement, one can see which side has been rotated and circumducted into a position of greater dominance.

1. *Shoulder Level Test.* Shrugging of the shoulder.[1] Because of the variations in the freedom of movement at the shoulder level, two types of response are possible: 1. the restricted type of movement is simply elevation of the shoulder or shoulders by abducting the clavicle. 2. the non-restricted type of movement involves simultaneously the clavicle, scapula and humerus: forward rotation and forward circumduction (*i. e.*, upward and forward movement of the clavicle and scapula) followed by some degree of medial rotation and medial circumduction (*i. e.*, abduction and flexion of the humerus). See figure 2.
2. *Elbow Level Test.* Crossing of the arms over the chest, resulting in the dominant arm being positioned superior to the non-dominant arm. The dominant side involves a greater degree of

[1] During the shoulder shrugging action, individual variations in the freedom of movement become apparent. The natural movement of the shoulder is from the non-dominant position and involves the humerus as well as the scapula and clavicle. When the movement is begun from the dominant position, one or more degrees of freedom are lost at the shoulder level, and the movement is restricted. For example, if the superficial back muscles are hypertonic, two degrees of freedom are lost; the natural movement will be abortive, so that shoulder elevation only is possible.

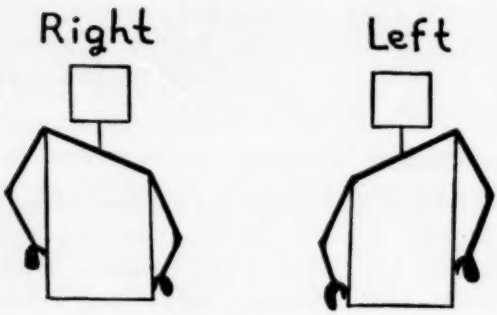

FIG. 2. SHOULDER DOMINANCE (1).

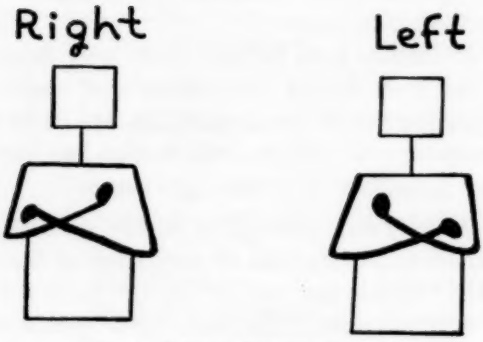

FIG. 3. ELBOW DOMINANCE (2).

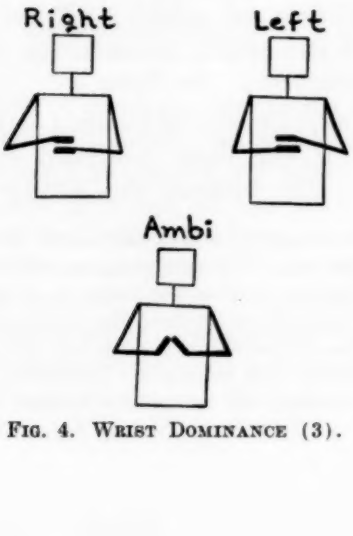

FIG. 4. WRIST DOMINANCE (3).

medial circumduction (flexion and adduction) of the elbow. Accompanying the elbow movements, there are minimal shoulder movements: medial rotation and medial circumduction of the humerus (Steindler, 1955). See figure 3.

3. *Wrist Level Test.* Clapping of the hands together, resulting in the dominant forearm being pronated (Darcus, 1951 and Steindler, 1955) and the wrist medially (radially) circumducted in two movements: extension (Anapol, 1932 and Steindler, 1955) and radial deviation. See figure 4.

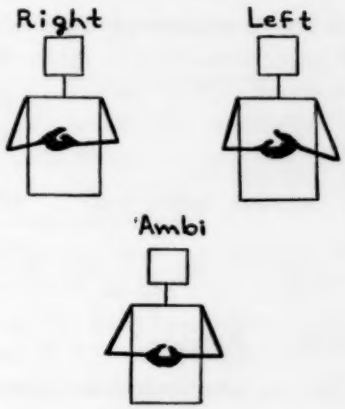

FIG. 5. THUMB DOMINANCE (4).

4. *Thumb Level Test.* (Lutz, 1908; Downey, 1926). Folding of the hands, with the fingers interlocked, so that the dominant thumb is positioned superior to the non-dominant thumb. The dominant action occurs at the carpo-metacarpal joint of the thumb and involves medial rotation accompanying medial circumduction in two movements: abduction and flexion. Rasch and Burke, 1960). See figure 5.

Subjects:

The population group studied represented freshmen medical students, from the University of Illinois and Chicago Medical School; and both medical and dental students from Loyola University, Chicago, during the period 1954-1959. They were predominantly male, averaging 21 years of age.

Each student was instructed by questionnaire to perform the simple self-test series; to observe and then to record his results. Students were

spot-checked for accuracy of their answers. A total of 943 questionnaires were counted for the handedness classification in table 2. However, since 26 of the subjects (2.6% of the total) did not complete the self-test series, their laterality dominance patterns could not be determined. A total of 917 questionnaires, with the self-test series completed, were used to construct the laterality patterns.

TABLE 2

Proportionate representation of handedness

SUBJECTS	#	%
Left-handed	92	9.8
Ambi-handed	124	13.1
Right-handed (heterozygous)	299	31.7
Right-handed (relatively homozygous)	428	45.4
Total	943	100.0

Handedness Classification of Subjects:

The subjects were divided into handedness groups by self-declaration:

1. *Left-handed group*—those who hold a pen in the left hand while writing and otherwise manipulate with the left hand.
2. *Ambi-handed group*—those who hold a pen in the right hand while writing, but manipulate objects such as balls and scissors with the left hand.
3. *Right-handed group*—those who hold a pen in the right hand while writing and otherwise manipulate with the right hand. These can be divided into relatively "homozygous" and "heterozygous" groups, depending upon the handedness of the immediate relatives for three generations. (a) The relatively homozygous group included those with known right-handed relatives only. (b) The heterozygous group included all those with known left- and ambi-handed relatives, in addition to right-handed relatives.

The three generations of relatives included: I. grandparents; II. parents and siblings of parents; III. siblings and cousins of subjects.

The proportional representation for each handedness group is summarized in table 2. Although 9.8% for left-handedness seems high, as compared with the low record of 4.3% for Ohio State University students by Chamberlain (1928), it compares well with the record of 8.6% by Karpinos and Grossman (1953) for 12,000 male Selective Service Registrants, ages 19–25. Chamberlain (1928) has shown that the percentages vary with the degree of homozygosity for handedness, so that with both parents left-handed, 46% of the offspring were also left-handed; but with neither parent left-handed, only 2.1% of the offsprings were left-handed.

In addition to the high percentage of left-handedness, there is a surprisingly high percentage of ambi-handedness, 13.1%, which has not increased over the years; Downey (1927) found mixed arm laterality of 10–15%; Leche, (1933) found 14% ambilaterality. Only one of the 943 subjects in the present study shows ambilateral dominance in writing. Koch, (1933) found by her scores 1/200 subjects who would show potential ambilateral equidominance.

The right-handed group, with no apparent functional ambilateral dominance represents 77.1% of the total subjects. Of these 45.4% are relatively homozygous for right-handedness, with no known left- or ambi-handed relatives; 31.7% are "heterozygous" for handedness and have known left- or ambi-handed relatives among the most recent three generations.

Sample Questionnaire:

The questionnaire used in this analysis of upper extremity dominance represents a part of a population survey relating handedness to familial patterns of body asymmetry.

A. Questions on handedness

1) Do you write with your: right hand ( ); left hand ( ); either hand ( ).

2) Do you handle a spoon with your: right hand ( ); left hand ( ); either hand ( ).

3) Were you left-handed or ambidextrous during childhood in reaching for toys ( ); throwing balls ( ); handling a spoon ( ); don't remember ( ).

4) Are any of the following relatives left-handed (L) or ambidextrous (A). If you don't know, use (?):

brothers ( )	sisters ( )
father ( )	mother ( )
paternal grandmother ( )	maternal grandmother
" grandfather ( )	" grandfather
" aunt	" aunt
" uncle	" uncle
" cousins	" cousins

B. Directions for self-testing of body asymmetry:

1) Rest your head on one shoulder and then on the other shoulder; which side feels more comfortable?
Left ( ) Right ( ) Both ( ) Neither ( ).

1a) (Alternate) If your answer to (1) is (Neither): Shrug your shoulders: which shoulder is elevated more freely?
Left ( ) Right ( ) Both ( ).

2) Cross your arms in front of your chest (your hands should be free): which arm lies superiorly? Left ( ) Right ( ).

3) Applaud with your hands: which hand is on top?
Left ( ) Right ( ) Neither ( ).

4) Fold your hands together (your hands should be free in order to do so): which thumb lies on top? Left ( ) Right ( ).

Validation of Questionnaire Method:

Subjects chosen for the study were mature students who are accustomed to carrying out written instructions with a maximum of accuracy. Any accidental errors would cancel out when large numbers are tabulated so that they could have no effect on the predominant tendency for the patterns in each handedness group. Their influence on the total dispersion of the patterns would be minimal and would show up primarily in the number of complete questionnaires. Out of a total of 943 subjects, there were 27 incomplete questionnaires, representing 2.6% of the total subjects. The ambi-handed showed the greatest number of incompletes, 3.2%; the left-handed showed the least number of incompletes, 2.1%. This slight, but interesting difference indicates that the ambi-handed are apparently the least sure of their laterality dominance and left a blank on the questionnaire; the left-handed are apparently most sure of

their laterality dominance and left fewer blanks on the questionnaire than any other group.

The total dispersion of the patterns was quite adequate to show the predominant tendency for each handedness group. If there were simply random distribution of the 81 (*i. e.*, 3^4) theoretical variations in pattern among the subjects, each pattern would then be represented by 11 examples. However, out of 81 theoretical variations in pattern, 30 variations were represented by from four to 85 subjects; 14 patterns were represented by one or two subjects; and 37 patterns were not represented at all.

As an additional check, the right-handed group, being especially large and totaling 708 members, was subdivided into two groups; the relatively homozygous right with 417 members and the heterozygous right with 291 members. The amazingly parallel results within 1–3% difference for the two groups, are represented by the frequency dispersion of the patterns in figure 6. This provides sufficient evidence for the accuracy of the self-test questionnaire.

Codifying of Tests into Dominance Patterns:

Starting from the theoretical absolute dominance values, the symbols (+) for right, (—) for left and (±) for ambi-lateral dominance were substituted for simplication; the numbers 1–4 were used for each of the four joint levels of the extremity; (1) shoulder, (2) elbow, (3) wrist, (4) thumb.

RESULTS

In analyzing the limb dominance patterns for each handedness group, one can compare:

1. the significant joint levels of dominance.
2. reinforcement or non-reinforcement of significant joint levels by other joint levels.
3. the grading of limb dominance by degrees from the finely graded pure right or left dominance patterns to the intermediate stages of mixed dominance patterns.

Joint Levels of Dominance

For each joint level, there may be either symmetry or asymmetry:

Symmetry. See table 3 for comparison of ambi-laterality in each handedness group, using the total number of subjects as a basis for

TABLE 3

Incidence of ambilaterality for each joint level

LEVELS	LEFT	AMBY	RIGHT (heterozygous)	RIGHT (relatively homozygous)
shoulder	47%	48%	41%	48%
elbow	—	1%	—	—
wrist	8%	7%	5%	5%
thumb	1%	—	—	—

Percentages were calculated from the total number of subjects in each handedness group.

TABLE 4

Joint levels of limb dominance

JOINT LEVELS	DOMINANCE SIGN	LEFT-HANDED	AMBI-HANDED	RIGHT-HANDED (heterozygous)	RIGHT-HANDED (relatively homozygous)
Shoulder	+1	22%	13%	39%	41%
Level	±1	64	65	61	59
	−1	14	22	0	0
Elbow	+2	32%	84%	62%	59%
Level	−2	68	16	38	41
Wrist	+3	10%	80%	100%	100%
Level	−3	90	20	0	0
Thumb	+4	32%	40%	59%	59%
Level	−4	68	60	41	41

The significant level of dominance for each handedness group was calculated by comparing the portion of each handedness group which best distinguishes itself by its homogeneity in pattern. There was a natural break in homogeneity following the seventh pattern of the frequency distribution. The more homogeneous portion represents the following percentage of each handedness group:

64% relatively homozygous right-handed group
59% heterozygous right-handed group
52% ambi-handed group
61% left-handed group

The remaining percentages are scattered among 37 variant patterns and therefore represent the more heterogeneous portion of each handedness group.

comparison. The shoulders show the maximum incidence of ambi-laterality (41–48%); the wrist only 5–8%; the elbow and thumb a mere 1%. The one truly ambidextrous individual who could write equally well with both limbs had ambi-laterality of both the wrist and thumb levels.

Asymmetry. In table 4, a comparison is made of all handedness groups at each of the four joint levels. Utilizing as a basis for comparison the top seven frequency patterns of each handedness group, one can distinguish more strikingly the differences between handedness groups in unilateral dominance.

At the *shoulder level,* there is such a high percentage of ambi-laterality, that unilateral dominance is not so conspicuous. The incidence of right shoulder dominance varies from 13–41%; the corresponding incidence for left shoulder dominance ranges from 0–22%. The relatively homozygous right-handed group shows the highest average of right dominance (41%) as well as the lowest average of left dominance (0%), at the shoulder level. The ambi-handed show just the opposite; the lowest average of right dominance (13%) and the highest average of left dominance (22%) at the shoulder level. Surprisingly, the left-handed show twice as many right dominant shoulders (22%) as left dominant shoulders (14%) with the precentages just the reverse of the ambi-handed. See table 4.

At the *elbow level,* the ambi-handed show the highest incidence of right dominance (84%). The left-handed show the highest incidence of left dominance (68%). There is only slight preferential right dominance for the right-handed groups: relatively homozygous (59%) and heterozygous (62%). See table 4.

At *wrist level,* both right-handed groups show 100% right dominance. The left-handed show 90–100% left dominance. The ambi-handed show, surprisingly, 80% right dominance. See table 4.

At the *thumb level,* there is more equal distribution of left and right dominance in all handedness groups. There is only moderate left dominance in both the left-handed (68%) and the ambi-handed (60%). There is only slight preferential right dominance in both right-handed groups (59%). See table 4.

Significant Levels of Dominance

By analyzing the data in table 4, the most significant joint level for each handedness group can be determined. The wrist level best differentiates the left-handed from the right-handed.

In the *right-handed groups,* the seven most frequently demonstrated patterns show a right dominant wrist (+ 3), with 100% incidence for both groups. In the *left-handed group,* six of the seven most frequently demonstrated patterns show a left dominant wrist (— 3), with 100% incidence, or with 90% incidence if all seven most frequently demonstrated patterns are considered.

For the *ambi-handed group,* there is no one level of dominance which outweighs all the rest; rather, it is a combination of levels with mixed dominance. It is revealing that four of the seven most frequently demonstrated patterns have in common with the right-handed groups, a right dominant wrist (+ 3), with 80% incidence. Even more significant than the wrist level is the high incidence of right dominance at the elbow level (+ 2) of 84%. Six of the seven most frequently demonstrated patterns are right dominant at the elbow level. This means that the opposite dominance, *i. e.,* left dominance, will usually show up at the shoulder or thumb levels in these patterns.

Degrees of Dominance

The most significant level of dominance, the wrist, may be reinforced by one or more other joint levels of dominance to determine wide variations in the degree of total limb dominance. If one temporarily excludes the very commonly ambilateral shoulder level, for convenience, and considers only the more distal levels: the elbow, the wrist and the thumb, one finds there are eight degrees of relative dominance possible. The variations range from the extreme all right dominant patterns (+ 2 + 3 + 4) to the extreme all left dominant patterns (— 2 — 3 — 4). Between the two extremes there are six intermediate degrees of mixed dominance. See figures 6-8 for a comparison of the frequency distributions in each handedness group.

(+ 2 + 3 + 4). This is the most frequently demonstrated right-handed combination, with an incidence of 22% for the heterozygous and 25% for the relatively homozygous groups. It is 3/3 right dominant from the elbow level to the thumb level. The remaining variations in pattern show an order of decreasing right dominance, with the more common patterns being 2/3 right dominant, inclusive of the wrist; and the least common patterns being only 1/3 right dominant, exclusive of the wrist.

(— 2 — 3 — 4). This is the most frequently demonstrated left-handed combination, with an incidence of 28%. It is 3/3 left dominant from the elbow level to the thumb level. The remaining variations in

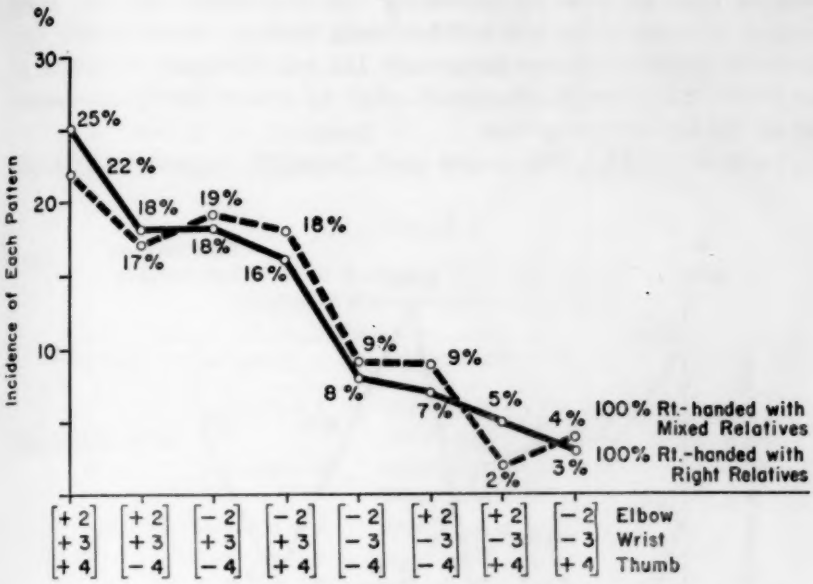

FIG. 6. COMPARISON OF % DISTRIBUTION FOR DOMINANT ARM PATTERNS BETWEEN RIGHT-HANDED WITH MIXED AND THOSE WITH ALL RIGHT-HANDED RELATIVES.

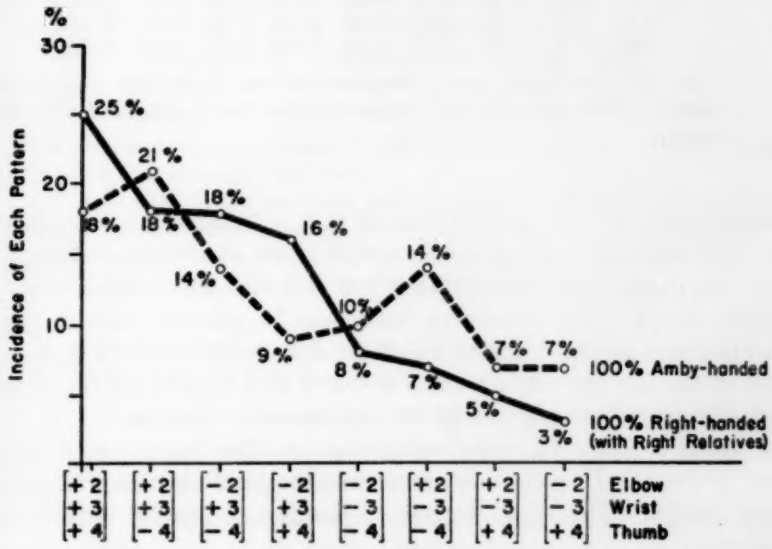

FIG. 7. COMPARISON OF % DISTRIBUTION FOR DOMINANT ARM PATTERNS BETWEEN RIGHT-HANDED (WITH RIGHT-HANDED RELATIVES) AND AMBI-HANDED SUBJECTS.

patterns show an order of decreasing left dominance, with the more common patterns being 2/3 left dominant, inclusive of the wrist; and the least common patterns being only 1/3 left dominant, exclusive of the wrist. Surprisingly, the $(+2-3+4)$ is consistently uncommon for all the handedness groups.

$(+2+3-4)$. This is the most frequently demonstrated ambi-

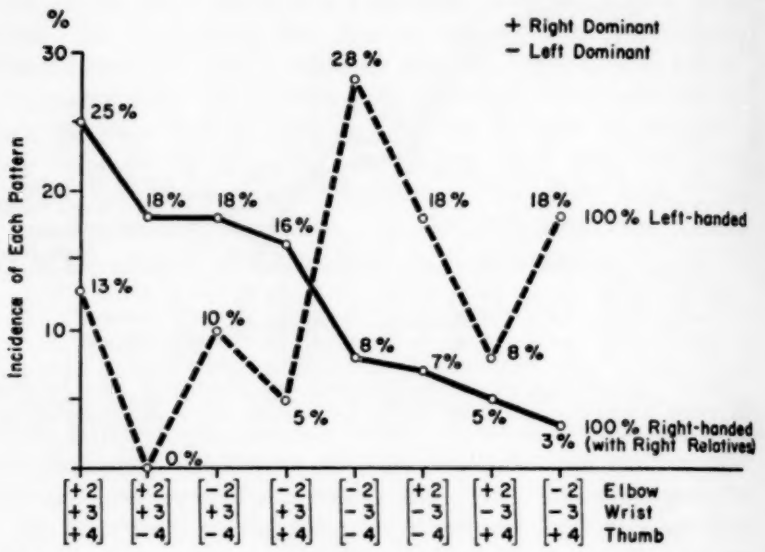

FIG. 8. COMPARISON OF % DISTRIBUTION FOR DOMINANT ARM PATTERNS BETWEEN LEFT-HANDED AND RIGHT-HANDED (WITH RIGHT-HANDED RELATIVES).

handed pattern, with an incidence of 21%. It shows a unique mixture of right and left dominance: it is 2/3 right dominant proximally (at the elbow and wrist levels) and 1/3 left dominant distally (at the thumb level). The remaining variations in patterns show a gradual reversal in dominance sign in which the more common patterns represent 2/3 of the top frequency pattern $(+2+3-4)$ and the less common patterns represent only 1/3 of the top frequency pattern.

Table 5 shows the relationship between the degrees of dominance and the relative frequency distribution of the patterns among the handedness groups. The eight degrees of dominance can be interpreted as follows:

I. 3/3 right dominance—all three joint levels tested (the elbow, (viii) the wrist and the thumb) are right dominant.

II. 2/3 right dominance—the two proximal joint levels (the elbow and the wrist) are right dominant but the more distal thumb level is left dominant.

III. 2/3 right dominance—the two more distal levels (the wrist and the thumb) are right diminant but the more proximal elbow level is left dominant.

IV. 1/3 right dominance—the wrist is right dominant, with alternation of left dominance both proximally at the elbow and distally at the thumb level.

V. 1/3 left dominance—the wrist is left dominant, with alternation of right dominance both proximally at the elbow and

TABLE 5

The eight degrees of limb dominance[1]
(Exclusive of the shoulder dominance)

DEGREES[2]	PATTERNS	HANDEDNESS GROUPS COMMONLY REPRESENTED
I (viii)	+2+3+4	Extreme Right-handed Pattern Ambi-handed Pattern
II	+2+3—4[3]	Maximum Ambi-handed Pattern Right-handed Pattern
III	—2+3+4	Right-handed Pattern
IV	—2+3—4	Right-handed Pattern Ambi-handed Pattern
V	+2—3+4[4]	Weak Left-handed Pattern Weak Ambi-handed Pattern
VI	+2—3—4	Left-handed Pattern Ambi-handed Pattern
VII	—2—3+4	Left-handed Pattern
VIII (i)	—2—3—4	Extreme Left-handed Pattern

[1] Each degree of limb dominance is determined by the reinforcement or non-reinforcement of significant joint levels.

[2] The upper case roman numerals indicate degrees of right dominance; the lower case numerals indicate degrees of left dominance.

[3] This pattern is absent among the left-handed group.

[4] This pattern shows the lowest incidence in all handedness groups.

distally at the thumb level. This is the most uncommon pattern in all the handedness groups.

VI. 2/3 left dominance—the two distal joint levels (the wrist and the thumb) are left dominant and the distal thumb level is right dominant.

VII. 2/3 left dominance—the two proximal joint levels (the elbow and the wrist) are left dominant and the proximal elbow level is right dominant.

VIII. 3/3 left dominance—all three joint levels tested (The elbow, (i) the wrist and the thumb) are left dominant.

There are several interesting complementary relationships that appear between the highest frequency and the lowest frequency patterns of one handedness group or between handedness groups

1. The top frequency pattern of the ambi-handed group (+ 2 + 3 — 4) is opposite in dominance sign to the low ranking pattern of the ambi-handed group, (— 2 — 3 + 4).
2. The top frequency pattern of both right-handed groups (+ 2 + 3 + 4) is opposite in sign to the top ranking pattern of the left-handed group (— 2 — 3 — 4).

Patterns of Limb Dominance

Pure and Mixed Dominance Patterns:

Only 9.8% of the total experimental group demonstrate a pattern of pure right dominance; and a mere 2.1% of the total group show a pattern of pure left dominance. The remaining 88.1% of the group present various patterns of mixed dominance. See table 6.

Total Variations:

If all degrees of freedom at each joint level are considered there are 81 theoretically possible patterns of limb dominance, *i. e.*, 3^4 variations. These patterns represent recombinations of three dominance types (+) right, (±) ambi, or (—) left, repeated at four separate joint levels: (1) shoulder, (2) elbow, (3) wrist, (4) thumb. However, some of the theoretical combinations of mixed dominance are quite rare, such as those involving ambilaterality at the elbow and the thumb levels and show up only in one or two individuals of the entire group (see table 8 for the variant patterns); 37 other patterns do not appear at all.

If the uncommon patterns are ignored, there are still 30 patterns represented by at least 4 and some by as many as 85 individuals. These patterns include: 1. all possible variations in unidominance; 2. all possible variations in mono-level ambi-dominance of the shoulder; 3. 3/8 of the possible variations in mono-level ambi-dominance of the wrist; 4. 3/4 of the possible variations in bi-level ambi-dominance of the shoulder and wrist.

TABLE 6

Pure dominance patterns

LEVEL	PURE RIGHT	MIXED	PURE LEFT
Shoulder	+ 1	( )	— 1
Elbow	+ 2	( )	— 2
Wrist	+ 3	( )	— 3
Thumb	+ 4	( )	— 4
Total %	9.8%	88.1%	2.1%

In table 7 the degrees of left dominance are opposite in sign to those of right dominance. They are represented by lower case roman numerals i to viii, in order to distinguish them from the degrees of right dominance as represented by upper case numerals I to VIII.

The seven top frequency patterns for each handedness group are italicized by rank number. These patterns represent the predominant tendency for each group. The predominant tendency for three of the handedness groups (including both right-handed groups and the ambi-handed group) is represented by degrees I to IV of right dominance; while the predominant tendency for the left-handed group is represented by degrees i to iii of left dominance.

For convenience, the key for interpreting the patterns is repeated: dominance signs (+) right, (±) ambi, (—) left; joint levels (1) shoulder, (2) elbow, (3) wrist, (4) thumb.

Percentages are based on 917 total subjects with completed self-test series: 417 right (relatively homozygous), 291 right (heterozygous), 120 ambi-, and 89 left-handed.

In table 7, all the variations in unidominance and ambi-dominance of the shoulder are ranked according to their theoretical degree of right or left dominance: the pure right dominant pattern (+ 1 + 2 + 3 + 4) being at the top of the list, and the pure left dominant pattern

TABLE 7

Common patterns of limb dominance

(Including all the possible variations in unidominance as well as in mono-level shoulder ambidominance)

DEGREE OF RIGHT DOMINANCE	PATTERN VARIATION	RIGHT-HANDED (homozygous)			RIGHT-HANDED (heterozygous)			AMBI-HANDED			LEFT-HANDED			TOTAL SUBJECTS		
		Rank	Freq.	%	Rank	Freq.	%	Rank	Freq.	%	Rank	Freq.	%	Rank	Freq.	%
I (viii)	+1+2+3+4	*1*	46	11.0	*1*	28	9.6	*4*	7	5.8	13	3	3.3	*2*	84	9.2
	±1+2+3+4	*2*	45	10.8	*2*	26	8.9	*2*	9	7.5	*5*	5	5.6	*1*	85	9.3
	−1+2+3+4	11	12	2.9	9	12	4.1	*5*	6	5.0	8	4	4.5	11	34	3.7
II	+1+2+3−4	*6*	28	6.7	*5*	22	7.5	*5*	6	5.0	24	0	—	*6*	56	6.1
	±1+2+3−4	*4*	37	8.9	*6*	19	6.5	*1*	13	10.8	24	0	—	*4*	69	7.5
	−1+2+3−4	16	8	1.9	13	8	2.7	13	3	2.5	24	0	—	17	19	2.1
III	+1−2+3+4	7	25	6.0	7	16	5.5	13	3	2.5	24	0	—	7	44	4.8
	±1−2+3+4	*5*	32	7.7	*4*	25	8.6	*5*	6	5.0	13	3	3.3	*5*	66	7.2
	−1−2+3+4	12	11	2.6	12	10	3.4	21	2	1.7	24	0	—	15	23	2.5
IV	+1−2+3−4	8	20	4.8	8	15	5.2	9	5	4.2	16	2	2.2	8	42	4.6
	±1−2+3−4	*3*	38	9.1	*2*	26	8.9	*2*	9	7.5	16	2	2.2	*3*	75	8.2
	−1−2+3−4	10	16	3.8	11	11	3.8	23	2	1.7	18	1	1.1	12	30	3.3
V	+1+2−3+4	21	4	1.0	26	1	0.3	13	3	2.5	8	4	4.5	22	12	1.3
	±1+2−3+4	15	9	2.2	18	4	1.4	21	2	1.7	8	4	4.5	17	19	2.1
	−1+2−3+4	24	2	0.5	26	1	0.3	24	1	0.8	24	0	—	26	4	0.4
VI	+1+2−3−4	12	11	2.6	15	7	2.4	13	3	2.5	8	4	4.5	13	25	2.7
	±1+2−3−4	14	10	2.4	9	12	4.1	9	5	4.2	*1*	11	12.3	9	38	4.1
	−1+2−3−4	20	5	1.1	18	4	1.4	*5*	6	5.0	18	1	1.1	20	16	1.7
VII	+1−2−3+4	27	1	0.3	18	4	1.4	13	3	2.5	*4*	6	6.7	21	14	1.5
	±1−2−3+4	18	7	1.7	18	4	1.4	11	4	3.3	*5*	5	5.6	16	20	2.2
	−1−2−3+4	36	0	—	26	1	0.3	34	0	—	8	4	4.5	28	5	0.5
VIII (i)	+1−2−3−4	16	8	1.9	13	8	2.7	13	3	2.5	*5*	5	5.6	14	24	2.6
	±1−2−3−4	9	17	4.1	16	6	2.1	11	4	3.3	*1*	11	12.3	9	38	4.1
	−1−2−3−4	24	2	0.5	16	6	2.1	13	3	2.5	*3*	7	7.9	19	18	2.0
Total			394			276			108			82			860	93.7+%

The rank numbers of the seven most frequently demonstrated patterns for each handedness group are italicized.

(— 1 — 2 — 3 — 4) being at the bottom of the list. The mixed variations between the pure dominance patterns are ranked according to their degree of right dominance, *i. e.*, according to the reinforcement or non-reinforcement of the most significant joint level, the wrist, in determining functional limb dominance. All the right dominant wrists are at the top of the list.

In comparing the pattern frequency distribution of each handedness group for degrees of right dominance, one finds: Degree I ranks first for both right-handed groups; Degrees I and II rank first for the ambi-handed group; Degree VIII (or i) ranks first for the left-handed group.

In comparing the overlap among the top ranking patterns, one finds that: for both right-handed groups, the seven top ranking patterns are identical, but not precisely of the same rank order; for the ambi-handed, five of the top seven patterns are identical to those of the right-handed group, but not of the same rank order; for the left-handed group, only one of the top seven patterns is identical to any of the top right-handed patterns.

Table 7 emphasizes the relationship of the ambi-handed group to the other handedness groups: the majority of the ambi-handed resemble the right-handed groups by demonstrating right dominant wrist patterns; only a minority resemble the left-handed group by demonstrating left dominant wrist patterns.

The Most Representative Patterns for Each Handedness Group

Top Ranking Left-handed Patterns

Variations	± 1 — 2 — 3 — 4	± 1 + 2 — 3 — 4	— 1 — 2 — 3 — 4
Incidence	(12.3%)	(12.3%)	(7.9%)

In these patterns, the wrist and thumb are both left dominant; the elbow is predominantly left dominant; the shoulder is predominantly ambi-dominant.

Top Ranking Ambi-handed Patterns

Variations	± 1 + 2 + 3 — 4	± 1 + 2 + 3 + 4	± 1 — 2 + 3 — 4
Incidence	(10.8%)	(7.5%)	(7.5%)

In these patterns, the wrist is right dominant; the thumb is predominantly left dominant; the elbow is predominantly right dominant; the shoulder is ambi-dominant.

Top Ranking Right-handed Patterns

Variations	± 1 + 2 + 3 + 4	+ 1 + 2 + 3 + 4	
Incidence	(10.8%)	(11.0%)	relatively homozygous
	(8.9%)	(9.6%)	heterozygous

The rank numbers of the seven most frequently demonstrated patterns for each handedness group are italicized.

TABLE 8

Variant patterns of limb dominance

(Including all the possible variants with mono-level wrist as well as with bi-level shoulder-wrist ambi-dominance)

LEVELS OF AMBIDOMINANCE	PATTERN VARIANTS	RIGHT-HANDED (homozygous)			RIGHT-HANDED (heterozygous)			AMBI-HANDED			LEFT-HANDED			TOTAL SUBJECTS		
		Rank	Freq.	%	Rank	Freq.	%	Rank	Freq.	%	Rank	Freq.	%	Rank	Freq.	%
Mono-level elbow	+1±2+3−4	27	1	0.3	26	1	0.3	34	0	—	24	0	—	31	2	0.2
	+1±2−3+4	36	0	—	34	0	—	24	1	0.8	24	0	—	35	1	0.1
wrist	+1+2±3+4	18	7	1.7	34	0	—	34	0	—	24	0	—	26	7	0.8
	+1+2±3−4	27	1	0.3	26	1	0.3	34	0	—	24	0	—	31	2	0.2
	+1−2±3+4	27	1	0.3	26	1	0.3	24	1	0.8	18	1	1.1	29	4	0.4
	+1−2±3−4	22	3	0.7	22	3	1.0	24	1	0.8	18	1	1.1	23	8	0.9
	−1+2±3+4	36	0	—	34	0	—	24	1	0.8	24	0	—	35	1	0.1
	−1+2±3−4	36	0	—	34	0	—	24	1	0.8	24	0	—	35	1	0.1
	−1−2±3+4	27	1	0.3	26	1	0.3	34	0	—	24	0	—	31	2	0.2
	−1−2±3−4	27	1	0.3	34	0	—	24	1	0.8	24	0	—	31	2	0.2
thumb	−1−2−3±4	36	0	—	26	1	0.3	34	0	—	24	0	—	35	1	0.1
Bi-level shoulder-elbow	±1±2+3−4	36	0	—	34	0	—	24	1	0.8	24	0	—	35	1	0.1
	±1±2−3−4	36	0	—	34	0	—	24	1	0.8	24	0	—	35	1	0.1
shoulder-wrist	±1+2±3+4	36	0	—	34	0	—	13	3	2.5	24	0	—	23	8	0.9
	±1+2±3−4	22	3	0.7	24	2	0.7	34	0	—	18	1	1.1	29	4	0.4
	±1−2±3+4	27	1	0.3	24	2	0.7	34	0	—	13	3	3.3	23	8	0.9
	±1−2±3−4	24	2	0.5	22	3	1.0	24	1	0.8	24	0	—	35	1	0.1
shoulder-thumb	±1+2+3±4	27	1	0.3	34	0	—	34	0	—	24	0	—	35	1	0.1
wrist-thumb	+1+2±3±4	36	0	—	34	0	—	34	0	—	18	1	1.1	35	1	0.1
Tri-level shoulder-elbow-wrist	±1±2±3+4	36	1	0.3	34	0	—	34	0	—	24	0	—	35	1	0.1
Total			23			15			12			7			57	6.1 %

In these patterns, the wrist, the elbow and thumb are all right dominant; the shoulder is equally right dominant or ambi-dominant.

The Signifiance of the Frequency Distribution of Patterns

Tables 7 and 8 represent a condensation of the total data. Table 7 represents a ranking of the more common variations each demonstrated by 4–85 individuals; table 8 repersents a ranking of the less common variants each demonstrated by 1–8 individuals for each pattern.

The percentages are meaningless unless identified with one particular population group, since it should be obvious that the percentage will vary (Chamberlain, 1928) according to the chance heterozygous assorting or homozygous clustering of the subjects. In this study 88.1% of the subjects show various mixtures of limb dominance, which seems surprisingly high. An analysis of the genetic composition of the population studied is in progress and will show the predominantly hybrid nature of the group and the percentages of genetic mixtures in relation to handedness groups and limb dominance patterns.

DISCUSSION

Laterality Determinants in Each Handedness Group:

Right dominance of the wrist level alone is significant as the indicator of "right-handedness" in writing. There is 100% right dominance of the wrist among the seven top-frequency dominance patterns (see table 4). The extent of reinforcement or non-reinforcement of the wrist level by other levels determines the degree of dominance, *i. e.*, whether the individual is strongly or mildly right dominant.

Contrariwise, left dominance of the wrist alone is necessary for effective "left handedness" in writing. There is 100%–90% left dominance of the wrist in the six or seven top-frequency dominance patterns (see table 4). The extent of reinforcement or non-reinforcement of the wrist level by other levels determines the degree of dominance, *i. e.*, whether the individual is strongly or mildly left dominant.

Mixed dominance is characteristic of the ambi-handed group. Most frequently the patterns show right dominance of the elbow and the wrist levels, with opposite left dominance of the shoulder and the thumb levels. By examination of the seven top-frequency patterns, it becomes clear that the ambi-handed represent varying degrees of mixed dominance, *i. e.*, mixtures of the two extreme dominance groups. See table 4 for

percentages based on the seven top-frequency patterns. A majority of 80% show right dominance at the wrist level, and are basically "right-handed" in writing. They more frequently than not show reinforcement of right dominance at the elbow level, which means they are strongly "right-handed" for writing. A minority of 20% show left dominance at the wrist level and are basically left-handed in writing. However, more frequently than not, these individuals with the left dominant wrist show non-reinforcement at the elbow level, which means they are only weakly "left-handed" for writing. Because right dominance at the elbow level occurs both as a reinforcement and as a non-reinforcement of wrist dominance, the seven top frequency patterns of the ambi-handed group show an incidence of 84% right elbow level dominance, which is the highest percentage dominance at that level for any handedness group (see table 4).

TABLE 9

Wrist reinforcement in the total ambi-handed group

WRIST DOMINANCE	ELBOW REINFORCEMENT	INCIDENCE
Right	Reinforced (+ 2 + 3)	39%
	Non- " (— 2 + 3)	24%
Left	Reinforced (— 2 — 3)	17%
	Non- " (+ 2 — 3)	20%
	Total Ambi-handed Group	100%

The Ambiguous Ambi-handed Group

The ambi-handed are defined as those who write with the right hand, but otherwise manipulate with their left hand. They represent a mixture of two opposite population groups: those with left dominant wrists and those with right dominant wrists (see table 9 for a comparison based on the total number of patterns).

Those individuals with right dominant wrists, representing 63% of the total ambi-handed, are basically right-dominant for writing. The wrist dominance is either reinforced at the elbow level (+ 2), or it is non-reinforced at the elbow level (— 2). There are more subjects showing reinforced patterns (+ 2 + 3) than non-reinforced patterns (— 2 + 3). This means that there is a large group of strongly right-handed individuals (39%), the (+ 2 + 3) group, among the ambi-handed.

Those individuals with left dominant wrists, representing 37% of the total ambi-handed, are basically left-dominant for writing. The wrist dominance is either reinforced at the elbow level (— 2) or it is non-reinforced at the elbow level (+ 2). There are not so many subjects showing reinforced patterns (— 2 — 3) as there are showing non-reinforced patterns (+ 2 — 3). This means that there is a relatively smaller group of strongly left-handed individuals (17%), representing the (— 2 — 3) pattern among the ambi-handed group.

The individuals with non-reinforcement of the wrist level, (— 2 + 3) or (+ 2 — 3), represent 44% of the total ambi-handed group. This group is not very strongly right or left dominant for writing.

Their top-frequency pattern of limb dominance, (+ 2 + 3 — 4), is not demonstrated among the left-handed subjects at all; however, it is very common among right-handed subjects who are not functionally ambi-handed. It becomes clear that dominance of both the right elbow and the right wrist are essential for effective writing with the right limb; and that individuals with the above pattern are essentially right-handed writers. Because of the left dominant thumb, individuals with such a pattern can effectively manipulate large objects such as bats, balls, hammers, and scissors with their left hand, *i. e.*, the left dominant thumb can be more effectively abducted before grasping than can the non-dominant right thumb. It also becomes obvious that the dominant position of abduction for the thumb is relatively less essential in writing than is the non-dominant position of adduction for the purpose of holding the pencil more firmly.

Of the entire ambi-handed group, there was only one subject who could write equally well with both hands. Surprisingly, his pattern showed ambilaterality for both the wrist and the thumb, which relatively few other people demonstrate as adults (+ 1 + 2 ± 3 ± 4). 50/917 subjects showed ambilaterality of the wrist, but only 3/917 showed ambilaterality of the thumb. Of all these ambidextrous combinations, there is but one pattern with bilevel symmetry of the wrist and the thumb, the 1/917 subjects.

The third most frequently demonstrated pattern among the ambi-handed group appears in equal numbers among the left-handed (+ 2 — 3 — 4); but it is relatively uncommon among the right-handed groups who show no functional ambi-laterality. Because of the lack of reinforcement of the left dominant wrist by a like elbow dominance, individuals showing this combination have a 50–50 chance of remaining functionally left-handed for writing, or being persuaded to transfer to

the right limb, such manipulations of cultural bias as writing. Because of the reinforcement of the left dominant thumb by the left dominant wrist, such an individual would be more than likely to continue to manipulate large objects with the left hand. A left dominant or symmetrical shoulder pattern would further reinforce the functional left- or ambi-handedness.

The above discussion of several ambi-handed patterns, in relation to functional dominance, demonstrates the significance of individual *joint levels* of *dominance* as well as the importance of *reinforcement* of each significant level by secondary levels, for effective purposeful movements.

SUMMARY

Dominant Positioning of the Limbs

"*Handedness*" might better be discussed in terms of the active or dominant positioning of the entire limb for manipulation of objects. This positioning involves a medial rotation and circumduction of the entire limb at four separate levels simultaneously: the shoulder, the elbow, the wrist, and the thumb; with partial duplication of the arc of movement at each level. These separate movements can be analyzed.

Levels

Shoulder

clavicle and scapula: Forward rotation; and forward circumduction.
humerus: Medial rotation; and medial circumduction.

Elbow: Medial rotation; and medial circumduction.

Wrist: Medial rotation; and medial circumduction.

Thumb: Medial rotation; and medial circumduction.

Using simple bilateral performance tests for each joint level, one can compare the left limb with the right limb for laterality differences in the degree of dominant positioning. Each level may show unilateral dominance or ambi-laterality. From the sum of the four *levels* of *dominance*, one can build a total pattern of limb dominance which can show either:

1. Pure dominance—either right or left—representing 10% and 2% of all subjects respectively.
2. Mixed dominance—representing 88% of all subjects.

Manipulation of Objects

For each type of purposeful manipulation, there is one essential joint level functionally dominant.

a. In writing—the dominant wrist is primarily involved; but secondarily the dominant elbow.
b. In manipulation of objects such as balls and scissors, the dominant thumb is involved; secondarily in reaching for the object, the dominant shoulder is involved.
c. In large movements of the entire extremity, such as in reaching for objects, batting, throwing, or in turning of the trunk, the dominant shoulder is involved.

The effectiveness of each manipulation is dependent upon the degree of reinforcement or non-reinforcement of each primary level by one or more secondary levels.

This analysis of limb patterns of dominance provides a phylogenetic functional basis for understanding the wide range of variations possible in the normal posturing of the upper extremities. It should convince one of the large but finite number of possibilities for mixed dominance of the limbs in a mixed population group; and of the minority representation of absolute dominance—either of the pure right or of the pure left form, in such a heterogeneous group.

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DISTRIBUTION OF BLOOD ANTIGENS AMONG GUATEMALAN INDIANS [1]

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ONE of the objectives in physical anthropology is to classify human beings into various groups according to their physical characteristics. As genetic traits have been identified, it has been increasingly possible to define a number of specific characteristics which vary with different populations. The serological blood groups are among the most useful for such definition, and numerous surveys have been made regarding their geographic distribution.

Thorough studies of the American Indians have been made and blood groups among Indian populations in South America have recently been reviewed by Salzano (1957) and Mourant (1954). Cabrera (1950) studied the Mayan Indians in Guatemala using the ABO system, and Aguirre, Tandon and Scrimshaw (1953) investigated the ABO, Rh and MN blood groups. A recent report by Matson and Swanson (1959) studied the distribution of blood groups from selected populations in Mexico, and the Mam and Quiché Indians in Guatemala, using the ABO, Rh, MNSs, P, Kell, Lewis, Duffy, Kidd and Diego systems.

The present paper summarizes the findings of a blood group survey started in 1958 which includes all the principal linguistic groups identified in Guatemala. It also presents some previously unpublished data collected and summarized by Aguirre, Tandon and Scrimshaw (1953).

MATERIALS AND METHODS

With the cooperation and technical advice of the Instituto Indigenista de Guatemala, a total of 260 individuals in 29 Indian communities, including the main linguistic groups, were surveyed. The map in figure 1, compiled by Goubaud Carrera (1946), identifies the geographic dis-

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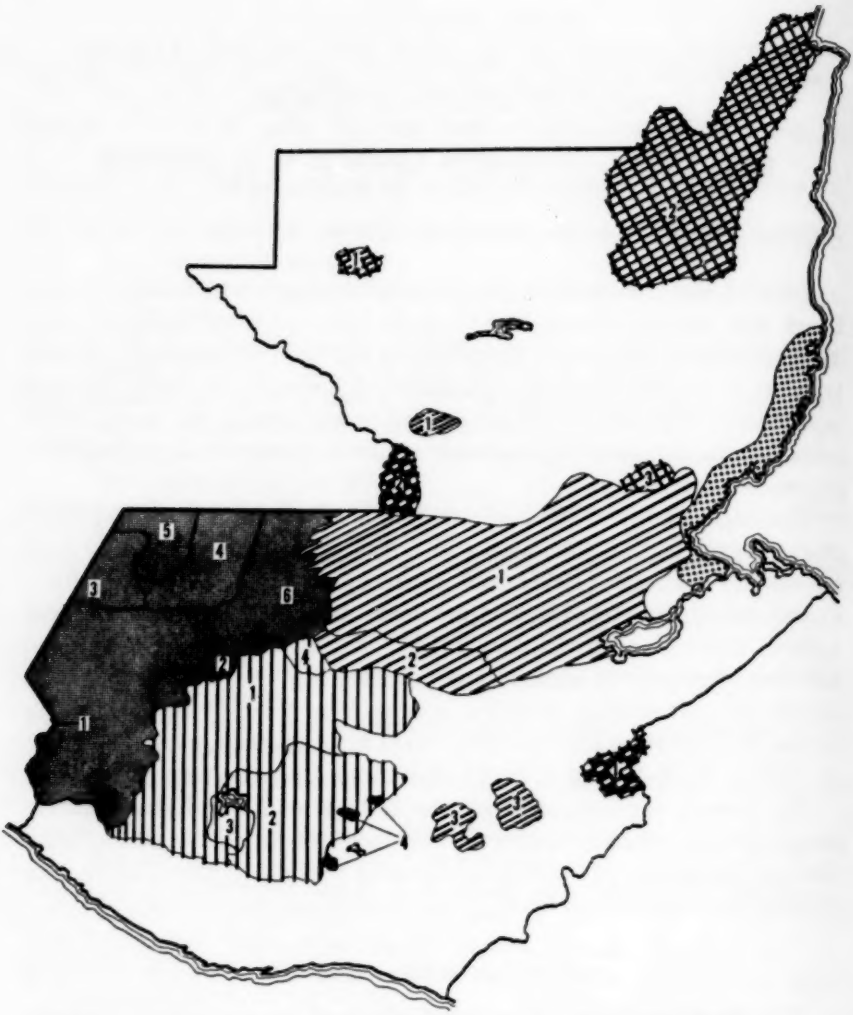

FIG. 1. MAP OF PRESENT INDIAN LINGUISTIC GROUPS OF GUATEMALA.

Compiled by: Lic. A. Goubaud Carrera and Prof. A. Arriaga (Goubaud Carrera, (1946).

QUICHE GROUP
1. QUICHE
2. CAKCHIQUEL
3. TZUTUJIL
4. USPANTECA
MAM GROUP
1. MAM
2. AGUACATECA
3. JACALTECA
4. KANJOBAL
5. CHUJ
6. IXIL
POCOMAM GROUP
1. KEKCHI
2. POCOMCHI
3. EASTERN POCOMAM
4. CENTRAL POCOMAM
CHOL GROUP
1. CHORTI
2. LACANDON CHOL
MAYAN GROUP
1. NORTHERN LACANDON
2. YUCATECO 3. MOPAN
CARIBBEAN GROUP
1. CARIBE ARAGUACO

tribution of the various linguistic groups, Quiché, Mam, Pocomam, Maya and Chol.

According to population, the main groups are the Quiché, Mam and Pocomam; the Chol and the Maya are now limited to a few localities. As shown in table 1, each of the main linguistic groups is divided into different subgroups. The Indian villages surveyed are presented in the same table, according to the linguistic groups and subgroups to which they belong. Most of the villages in this survey were studied previously by Aguirre, Tandon and Scrimshaw (1953) for the ABO, Rh and MN systems. The dotted area locates the Carib group which consists mainly of Negroes and mixtures of white with Negro (mulatto) or Indian with Negro (Red Carib). In the remaining white area there is no predominating linguistic group and the majority of people speak Spanish.

In each Indian community an effort was made to obtain ten blood samples from selected, non-related Indians. The field workers of the Instituto Indigenista were well acquainted with the communities, spoke the local dialects and selected the candidates on the following basis: a) acceptance of status as an Indian, b) wearing of their typical native costumes, c) speaking Indian dialect, and d) in some communities, possessing an Indian last name. This last criterion was not always possible to follow since several Indian communities have already translated the old Indian names into Spanish or changed the last name to one of Spanish origin.

The blood group systems studied were: ABO (119 cases), Rh (40), MNSs (260), P (260), Kell (260), Duffy (260), Diego (260), Kidd (220). The typing sera were obtained from different commercial firms and the techniques employed were those recommended by them.[3] The anti Di^a serum was kindly provided by Dr. Miguel Layrisse of Venezuela, and the technique recommended by him (personal communication) was used.

RESULTS AND DISCUSSION

Rh Blood Group: Tables 2 and 3, which include previously unpublished data of work by Aguirre, Tandon and Scrimshaw (1953), show the phenotypes and the expected genotype frequencies, as well as the

[3] Sera from Dade Reagents, Inc., Miami, Fla., were used for typing ABO, P, Kell, Kidd, Diego, Duffy and S blood groups. Sera from Ortho Pharmaceutical Corp., Raritan, N. J., were used for typing the ABO, Rh and MNS blood groups (part of them were kindly supplied by the corporation). Sera from Knickerbocker Blood Bank, New York, N. Y., were used for typing the P, Kell, Kidd, Duffy and S blood groups.

TABLE 1

Villages surveyed and linguistic groups to which they belong

MAIN LINGUISTIC GROUP	SUBGROUP	VILLAGE	CASES	TOTAL
Chol	Chortí	Olopa	10	
		Jocotán	10	
		Camotán	10	
	Lacandón Chol		0	30
Pocomam	Kekchí	Cobán	11	
		Carchá	12	
		Lanquín	11	
		Chaal	1	
		Chamelco	2	
		Senahú	1	
		Cahabón	2	
	Pocomchí		0	
	Pocomam Oriental	San Luis Jilotepeque	10	
		San Pedro Pinula	10	
	Pocomam Central	Palín	10	
		Chinautla	10	80
Quiché	Quiché	Nahualá	10	
		Santa Lucía Utatlán	10	
		San Mateo	10	
		San Andrés Xecul	10	
	Cakchiquel	Sumpango	10	
		Santa María de Jesús	10	
		Patzún	10	
		Comalapa	10	
	Tzutujil		0	
	Uspanteca		0	80
Mam	Mam	San Juan Ostuncalco	10	
		Concepción CH	10	
		San Pedro Sacatepéquez	10	
		San Sebastián Huehuetenango	10	
	Aguacateca	Aguacatán	10	
	Jacalteca		0	
	Kanjobal		0	
	Chuj			
	Ixil	Santa María Nebaj	10	
		Chajul	10	70
			0	
Maya	Lacandón del Norte			
	Yucateco		0	
	Mopán		0	0
Caribe	Caribe Araguaco		0	0
		Total No. of cases		260

expected chromosome and gene frequencies. The Guatemalan Indians have a high chromosome frequency of CDe (R^1) and cDE (R^2), and a low relative frequency of cdE (r''), CdE (r^y) and Cde (r'). According to Mourant (1954), "the American Indians and Eskimos have high and

TABLE 2

The Rh blood group

	PHENOTYPE FREQUENCIES		
	NO.	OBSERVED	EXPECTED
ccde	1	0.0040	0.0009
ccDe	3	0.0120	0.0701
ccDE	35	0.1480	0.0949
CcDe	12	0.0510	0.2028
CcdE	7	0.0300	0.0037
CcDE	94	0.4000	0.2743
CCde	2	0.0090	0.0019
CCDe	63	0.2680	0.1466
CCDE	17	0.0720	0.1981
CCdE	1	0.0040	0.0026
ccdE	0	0.0000	0.0013
Ccde	0	0.0000	0.0027
Total	235	0.9980	0.9999

TABLE 3

The Rh blood group

EXPECTED CHROMOSOME FREQUENCIES		EXPECTED GENE FREQUENCIES	
cde	0.063	C	0.591
Cde	0.051	c	0.409
cdE	0.000	D	0.886
cDe	0.063	d	0.114
CDe	0.475	E	0.348
CDE	0.065	e	0.652
cDE	0.283		
CdE	0.000		

approximately equal frequencies of CDe and cDE; cDe and CDE are often present but d in any combination is infrequent or absent." The high frequency of cDE appears to be confined to the American Indians, Polynesians and Eastern Asiatics.

ABO Blood Group: Table 2, which includes previously unpublished data from our Institute, shows that, on the whole, the Guatemalan Indians are predominantly group O people. The O gene frequency is similar to that found in previous studies of Guatemalan Indians by Cabrera (1950) and Matson and Swanson (1959), but it differs in the Black Caribs of Livingston, Guatemala, according to a previous study by Scrimshaw, Méndez, Flores, Guzmán and León (1961) which showed gene frequencies of .723, .137 and .150 for the O, A and B genes, respectively. The presence of some A and B genes among certain Indians is probably due to a degree of racial crossing. According to Mourant (1954) it is possible that the South and Central American Indians may have been exclusively group O before the coming of the Europeans.

MNSs Blood Group: Table 5 shows the results obtained in the MNSs group. The Ms chromosome is the most common and ranges from .4951 in the Quiché group to .7695 in the Chol; this chromosome is lower in both Europeans and African Negroes. In some aboriginal Malays and Chinese (Mourant, 1954), it is quite similar to that observed in the present study. However, differences are not frequent among American aborigines (Mourant, 1954; Matson and Swanson, 1959) except in a few tribes (Sandoval and Henckel, 1954; Matson, Koch and Levine, 1954; Pantin and Kallsen, 1953).

In our study, the Chol and Mam groups have higher Ms chromosome frequencies than the other groups, and there is a significant difference ($P < 0.05$) in the relative frequency of this Ms chromosome when the combined Pocomam-Quiché are compared with the combined Chol-Mam groups.

P Blood Group: Only anti P_1 (anti P) sera were used in testing this system. As shown in table 6, the P_1 gene frequency varies from .3239 in the Mam to .5170 in the Chol group with a total average of .4282. The P system has not been studied before in the Guatemalan Indians. The frequency of the P_1 in the Mayan Indians from Mexico is similar (Matson and Swanson, 1959).

The frequency of the two genes among many other races does not vary greatly from that of the Guatemalan Indians. Apparently the P_1 is slightly lower in some Asiatic people (Lehmann and Cutbush, 1952) and higher in African (Barnicot and Lawler, 1953; Henningsen, 1950; Shapiro, 1953) and American Negroes (Miller, Tannor and Hsu, 1950). The frequency of the P_1 gene is significantly less ($P < 0.05$) among the Mam groups than among the other three Indian groups, which are similar.

TABLE 4

The ABO blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES								EXPECTED GENE FREQUENCIES		
		A		B		AB		O		A	B	O
		No.	Frequency	No.	Frequency	No.	Frequency	No.	Frequency			
Chol	24	3	.1250	0	.0000	0	.0000	21	.8750	.0646	.0000	.9354
Pocomam	132	7	.0530	4	.0303	0	.0000	121	.9167	.0269	.0153	.9578
Quiché	129	1	.0078	1	.0078	0	.0000	127	.9844	.0039	.0039	.9922
Mam	70	3	.0429	2	.0286	0	.0000	65	.9285	.0217	.0144	.9639
Total	355	14	.0394	7	.0197	0	.0000	334	.9409	.0199	.0099	.9702

TABLE 5

The MNSs blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES												EXPECTED CHROMOSOME FREQUENCIES			
		MMS		MNS		MNs		MMs		NNS		NNs		MS	NS	Ns	Ms
		No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.	No.	Freq.				
Chol	30	2	.0667	2	.0667	6	.2000	18	.6000	1	.0333	1	.0333	.0306	.0566	.1433	.7695
Pocomam	80	24	.3000	11	.1375	19	.2375	19	.2375	3	.0375	4	.0500	.2207	.0547	.2203	.5043
Quiché	80	22	.2750	18	.2250	14	.1750	21	.2625	2	.0250	3	.0375	.2424	.0684	.1941	.4951
Mam	70	6	.0857	9	.1285	22	.3143	26	.3714	4	.0571	3	.0428	.0434	.1031	.2183	.6352
Total	260	54	.2077	40	.1538	61	.2346	84	.3231	10	.0385	11	.0423	.1521	.0733	.2017	.5729

Kidd Blood Group: The commercial anti Jk^a sera was used in testing for the Kidd (Jk^a) antigen. Only a weak agglutination was observed with the commercial antisera, although positive and negative controls were satisfactory. The total average of the Jk^a gene is .3091 as shown in table 7 which in itself is lower than the ones described elsewhere

TABLE 6

The P blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES		EXPECTED GENE FREQUENCIES	
		P + $P_1P_1 + P_1P_2$	P — P_2P_2	P_1	P_2
Chol					
No.	30	23	7		
Frequency		0.7667	0.2333	0.5170	0.4830
Pocomam					
No.	80	59	21		
Frequency		0.7375	0.2625	0.4876	0.5124
Quiché					
No.	80	55	25		
Frequency		0.6875	0.3125	0.4409	0.5591
Mam					
No.	70	38	32		
Frequency		0.5429	0.4571	0.3239	0.6761
Total					
No.	260	175	85		
Frequency		0.6731	0.3269	0.4282	0.5718

(Mourant, 1954) with the exception of a group of Chinese studied in New York (Rosenfield *et al.*, 1953) which show a frequency of .3103. The Jk^a gene is quite similar among the Chol, Pocomam and Quiché groups, but is lower ($P < 0.01$) in the Mam group.

Matson and Swanson (1959) reported higher values among Guatemalan Indians for the Jk^a gene. The difference could be a result of some false negatives due to the weak agglutination observed with the commercial antisera used in our work.

Kell Blood Group: Nearly all the Indians, with the exception of one case in the Chol group, were Kell negative (see table 8). These findings agree with values reported by others in aboriginal populations from America (Matson and Swanson, 1959; Chown and Lewis, 1953), with the exception of some Brazilian Indians reported by Pantin and Junqueira (1952) who demonstrated relatively high K frequency of .1241. The presence of K+ genes in one of our cases is due presumably to some Caucasian admixture.

TABLE 7

The Kidd (Jk^a) blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES		EXPECTED GENE FREQUENCIES	
		Jk^a+ $Jk^aJk^a + Jk^aJk^b$	Jk^a- Jk^bJk^b	Jk^a	Jk^b
Chol					
No.	30	19	11		
Frequency		0.6333	0.3667	0.3945	0.6055
Pocomam					
No.	40	25	15		
Frequency		0.6250	0.3750	0.3876	0.6124
Quiché					
No.	80	48	32		
Frequency		0.6000	0.4000	0.3675	0.6325
Mam					
No.	70	23	47		
Frequency		0.3286	0.6714	0.1806	0.8194
Total					
No.	220	115	105		
Frequency		0.5227	0.4773	0.3091	0.6909

Duffy Blood Group: As shown in table 9, there is no difference in the distribution of the Duffy system among the four Indian groups studied. The Fy^a gene was present in the total population with an average relative frequency of .5443. This value is lower than the one reported by Matson and Swanson (1959) in Mayan Indians and higher than the

values reported for European whites (Race and Sanger, 1952) and African Negroes (Shapiro, 1953). The latter show very low values.

Diego Blood Group: The anti Di^a serum was obtained through Dr. M. Layrisse from the Banco de Sangre, Caracas, Venezuela. The Di^a gene frequency, as shown in table 10 is higher among the Chol group (.2929) and lower among the Pocomam (.1126) with an average relative

TABLE 8

The Kell blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES		EXPECTED GENE FREQUENCIES	
		K + KK + Kk	K — kk	K	k
Chol					
No.	30	1	29		
Frequency		0.0333	0.9667	0.0168	0.9832
Pocomam					
No.	80	0	80		
Frequency		0.0000	1.0000	0.0000	1.0000
Quiché					
No.	80	0	80		
Frequency		0.0000	1.0000	0.0000	1.0000
Mam					
No.	70	0	70		
Frequency		0.0000	1.0000	0.0000	1.0000
Total					
No.	260	1	259		
Frequency		0.0038	0.9962	0.0019	0.9981

frequency of .1680. The Quiché and Mam groups show intermediate values. The Diego factor studied previously in Guatemala (Matson and Swanson, 1959) in 46 cases from the Quiché group, showed a gene frequency of .1739 which is quite similar to the value found in the present study. There is a significant difference ($P < 0.05$) between the Chol and the other three groups.

The Di^a frequency reported for the Chol group is probably one of the highest found up to now and is very close to that of the Brazilian

Caingangue and Carajá Indians (Layrisse and Wilbert, 1960). This factor, which has been well studied by these authors, has not been found among the Caucasian whites and African Negroes but is present in the American Indians and Asiatic Mongoloids. As suggested by them, the

TABLE 9

The Duffy (Fy^a) blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES		EXPECTED GENE FREQUENCIES	
		Fy^a + Fy^{a}Fy^a + Fy^{a}Fy^b	Fy^a — Fy^{b}Fy^b	Fy^a	Fy^b
Chol					
No.	30	26	4		
Frequency		0.8667	0.1333	0.6349	0.3651
Pocomam					
No.	80	63	17		
Frequency		0.7875	0.2125	0.5390	0.4610
Quiché					
No.	80	65	15		
Frequency		0.8125	0.1875	0.5669	0.4331
Mam					
No.	70	52	18		
Frequency		0.7429	0.2571	0.4929	0.5071
Total					
No.	260	206	54		
Frequency		0.7923	0.2077	0.5443	0.4557

presence of this gene in the American aborigine could be considered as evidence of a probable genetic relationship with Asiatic Mongoloids.

SUMMARY

Two hundred and sixty non-related Guatemalan Indians of Mayan extraction were selected according to the four major linguistic groups prevalent in the area; namely, Chol, Pocomam, Quiché and Mam, and studied for the distribution of the blood factors ABO, MNSs, P, Kell, Kidd, Duffy and Diego. The number tested, as well as the phenotype

and gene frequencies for each group, are presented. In the particular case of the ABO and Rh blood groups, some previously unpublished data are also included. Tables for the phenotype, genotype, chromosome and gene frequencies observed are presented, as well as a map localizing the surveyed areas.

TABLE 10

The Diego (Di^a) blood group

GROUPS OF POPULATION	NUMBER STUDIED	OBSERVED PHENOTYPE FREQUENCIES		EXPECTED GENE FREQUENCIES	
		Di^a + $Di^aDi^a + Di^aDi^b$	Di^a — Di^bDi^b	Di^a	Di^b
Chol					
No.	30	15	15		
Frequency		0.5000	0.5000	0.2929	0.7071
Pocomam					
No.	80	17	73		
Frequency		0.2125	0.7875	0.1126	0.8874
Quiché					
No.	80	25	55		
Frequency		0.3125	0.6875	0.1708	0.8292
Mam					
No.	70	23	47		
Frequency		0.3286	0.6714	0.1806	0.8194
Total					
No.	260	80	180		
Frequency		0.3077	0.6923	0.1680	0.8320

Most common, over-all gene and chromosome frequencies found were: CDe .475; O .9702; Ms .5729; P_2 .5718; Jk^b .6909; k .9981; Fy^a .5443; and Di^b .8320.

In the particular case of the Mam group, some unexpected differences were observed in the P, Kidd and MNSs groups. It is possible that this group is genetically different from the others, but further studies are necessary to establish this fact.

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CURRENT HEIGHT AND WEIGHT OF YOUTHS OF MILITARY AGE

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THE analysis relates to all youths of military age and to those youths who were inducted into military service. The findings are by age and race. They are compared with similar findings of former years, specifically with those of World Wars I and II.

SOURCE AND NATURE OF DATA AND PERIOD COVERED

The height and weight data used in this study pertain to Selective Service registrants who were examined by the Armed Forces examining stations during the 21-month period from January 1957 through September 1958. The examining stations are required to submit to the Office of The Surgeon General, Department of the Army, a copy of the medical examination report ("Report of Medical Examination," Standard Form 88) of each registrant disqualified by them for military service and of each qualified registrant inducted into the Army.

During this period, 50% of the submitted medical examination reports were selected and coded for this and other studies (Karpinos, 1960). The selection of the sample was made by taking all reports of the disqualified registrants whose Selective Service number assigned to them by their local boards ended in an odd digit, and all reports of the inducted registrants whose Armed Forces service number so ended.

MEASURING PROCEDURES

Height and weight are obtained routinely on the medical examinations and recorded on the medical reports (Standard Form 88).

With respect to measuring height, the standing instructions have been as follows:

> "Use a board at least 2 inches wide by 80 inches long, placed vertically, and carefully graduated to ¼ inch between 58 inches from the floor and the top end. Obtain the height by placing vertically, in firm contact with the top of the head, against the measuring rod an accurately squared board of about 6 by 6 by 2 inches, best perma-

nently attached to graduated board by a long cord. The individual should stand erect with back to the graduated board, eyes straight to the front" (Department of the Army, 1948).

The instructions further provide that the examinee's height be recorded in inches, to the nearest quarter inch, without shoes (Department of the Army, 1956).

With respect to examinee's weight, the instructions state that it be taken with the clothing removed, on a standard set of scales known to be correct, and recorded to the nearest pound (Department of the Army, 1956).

These instructions obviously provide for sufficient accuracy. Of course, greater accuracy, but limited in scope of investigation, is attained in height-weight studies specifically undertaken for that purpose. The equipment, or devices, used in such instances are more accurate and the measurements are more exact (Stoudt *et al.*, 1960).

STANDARDS OF ACCEPTABILITY FOR MILITARY SERVICE

Inasmuch as the height and weight standards established by the Armed Forces affect the height and weight of examinees found fit for military service, these standards are briefly stated here. Height-weight tables are published in Army regulations which specify the minimum and maximum acceptable weight for a given height. Examinees whose height and weight fall within the height-weight limits are obviously acceptable, unless disqualified for other (moral, mental, or medical) reasons. Examinees whose weights exceed the standards indicated for the particular heights may be accepted, provided the weights are not so excessive as to interfere with military training.

The minimum acceptable height for military service during this period was 60 inches, and the maximum height 78 inches. The required minimum weight was 105 pounds, except for Filipinos, Puerto Ricans, and individuals of oriental descent, for whom the minimum acceptable weight was 101 pounds.

As a general policy, the regulations state that the examining physicians use discretion in accepting persons whose height or weight deviates from the standards. Proper allowances are permitted in such cases, if, in the opinion of the examining physician, the deviations are temporary and correctable with proper food and physical training (Department of the Army, 1956).

DATA USED

The analysis deals with examinees of known height and weight. Height and weight were reported for practically all inductees. Among the disqualified examinees, the proportion for whom no height or weight was reported, ranged between 0.5 and 1.7%.

The study included some 273,000 medical reports, relating to examinees within the age range of 17 through 25. A distribution of these

TABLE 1

Distribution of the Medical Examination Reports included in the analysis, relating to registrants examined for military service, by age, race, and military qualification of examinees

(January 1957 through September 1958) [1]

AGE (AS OF LAST LAST BIRTHDAY)	NUMBER INCLUDED IN THE ANALYSIS			
	WHITE		NEGRO	
	Disqualified (1)	Inducted (2)	Disqualified (3)	Inducted (4)
Total (17–25)	109,737	111,622	37,176	14,362
17	525	1,035	282	208
18	3,698	9,200	2,612	1,935
19	3,132	11,323	1,930	1,723
20	2,241	8,344	1,207	1,120
21	23,871	17,263	8,314	1,980
22	65,351	56,113	21,350	6,529
23	6,507	5,448	965	613
24	2,571	1,844	305	147
25	1,841	1,052	211	107

[1] Source: 50 percent sample of Standard Forms 88 ("Report of Medical Examination") received in the Office of The Surgeon General, Department of the Army, relating to registrants who were disqualified for military service and to those who were found fit for military service and inducted into the Army.

medical reports, by age, race, and military qualification of the examinees (disqualified or inducted) is shown in table 1.

Age is given as of last birthday. The term "White" used in the racial classification refers to non-Negro. As may be seen from table 1, the majority of the examinees fell within the 22–23 age group. The mean ages were: white examinees—22.0 years; white inductees—21.6 years; Negro examinees—21.6 years; Negro inductees—21.3 years; total

(white and Negro) examinees—21.9 years; total (white and Negro) inductees—21.5 years. (See figure 1 for distribution of examinees by age and race.)

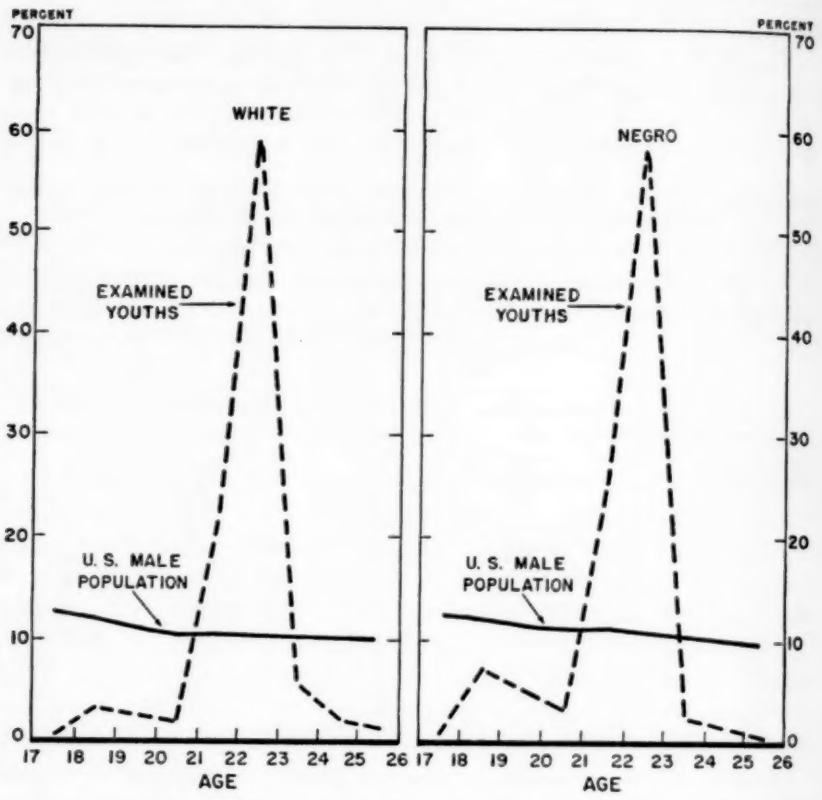

FIG. 1. PERCENT DISTRIBUTION OF YOUTHS, 17–25 YEARS OF AGE, EXAMINED FOR MILITARY SERVICE IN 1957–1958 AND OF THE 1960 TOTAL MALE POPULATION WITHIN THE SAME AGE RANGE, BY SINGLE YEARS OF AGE AND RACE.

The obtained height and weight data were cross-tabulated: height by single inches against weights in 5-pound intervals. The disqualified and inducted subclasses were tabulated separately. The cross-tabulations within each subclass were by race and by single years of age within race, in order to determine both race and age differentials.

MEAN HEIGHTS AND WEIGHTS

Age-Specific Values. The derived mean height and weight values and their standard deviations are shown in table 2. These values are

TABLE 2

Mean heights and weights of registrants examined for military service and standard deviation of these means, by age, race, and military qualification of the examinees

(January 1957 through September 1958) [1]

AGE (AS OF LAST BIRTHDAY) AND MILITARY QUALIFICATION OF EXAMINEES	HEIGHT (INCHES)				WEIGHT (POUNDS)			
	White		Negro		White		Negro	
	Mean (1)	S.D. (2)	Mean (3)	S.D. (4)	Mean (5)	S.D. (6)	Mean (7)	S.D. (8)
Inducted								
17	67.61	2.55	68.00	2.72	140.81	22.08	141.61	18.30
18	68.24	2.68	68.29	2.69	147.62	24.03	145.29	19.85
19	68.71	2.66	68.59	2.65	154.79	24.89	150.40	21.67
20	68.89	2.68	68.60	2.78	156.64	24.41	151.90	22.58
21	68.99	2.63	68.88	2.62	158.24	24.08	155.32	21.63
22	69.02	2.64	68.95	2.66	159.50	24.44	156.73	22.30
23	68.73	2.72	68.73	2.64	158.64	25.06	157.81	24.92
24	68.32	2.76	68.88	2.46	156.14	23.77	153.76	21.35
25	68.22	2.71	68.32	3.07	157.15	25.41	155.37	21.47
Total (17–25)	68.92	2.66	68.84	2.67	158.37	24.53	155.00	22.25
Examined [2]								
17	67.41	2.69	67.85	2.72	139.19	23.38	141.23	19.10
18	67.93	2.78	68.14	2.68	145.30	25.21	144.33	20.22
19	68.35	2.84	68.37	2.71	151.81	26.88	148.69	21.61
20	68.59	2.83	68.40	2.78	154.05	26.14	150.80	22.77
21	68.82	2.75	68.66	2.70	157.69	26.95	154.05	22.55
22	68.88	2.76	68.71	2.72	159.35	27.19	155.91	23.26
23	68.50	2.85	68.49	2.78	157.60	27.12	156.23	25.49
24	68.11	2.89	68.52	2.58	155.44	25.23	153.48	23.15
25	68.01	2.83	68.11	3.00	155.78	25.82	154.24	24.80
Total (17–25)	68.75	2.78	68.61	2.74	157.84	27.00	154.00	23.03
Total—Weighted by 1960 U. S. Male Population Ages (17 through 25), by Race [3]	68.26	2.83	68.35	2.76	152.27	26.77	150.65	23.10

[1] See footnote to Table 1. S.D. = Standard Deviation.

[2] Computed by "weighting" the height and weight values, obtained separately for the inductees and disqualified registrants. (See "Appendix: Technical Notes," 1.)

[3] Derived from the height and weight values by single years of age for "Examined," by weighting these values proportionately to the distribution of the 1960 U. S. male population, ages (17–25), by single years of age and by race. (See "Appendix: Technical Notes," 2.)

presented for "inducted" and "examined" registrants. The values of the "examined" registrants were computed from the separate tabulations of "inducted" and "disqualified" registrants by "weighting" these data, necessitated by the disproportionate representation of the subclasses: inducted in relation to disqualified. (See "Appendix: Technical Notes," 1.)

The data indicate maximum height at age 22. This holds for both white and Negro inducted and examined registrants. The maximum heights were: white inducted—69.02 inches, white examined—68.88 inches; Negro inducted—68.95 inches, Negro examined—68.71 inches. Beyond age 22, the heights are lower. Such decreases have been ordinarily interpreted as reflecting certain physiologic changes after the individuals have reached skeletal maturity, associated with changing postural habits as age increases. But such an exclusive interpretation neglects to take into account the secular trend of increasing height as a contributing (statistical) factor, since the older ages were originally of lower stature.

The standard deviations of height show a general trend of increasing variability with age (table 2).

With respect to weight, the data indicate for white inductees and examinees maximum weights at age 22: inductees—159.50 pounds, and examinees—159.35 pounds; for Negro inductees and examinees maximum weights are indicated at age 23: inductees—157.81 pounds, and examinees—156.23 pounds.

The age-specific height and weight values of "Examined" (table 2) may be taken as representative of all youths, though they exclude youths exempt from military service. The exclusion of this group of youths could not have appreciably affected the derived age-specific height and weight values, since there is no basis to assume that the height and weight of the exempt youths would differ materially from those of youths examined. Moreover, compared with the number of youths examined, the number of exempt youths has been relatively too small to have a distorting effect, even if certain differences do exist.

Racial Differences. In general, there seems to be practically no difference between the current height of white and Negro youths. However, the white youths appear to be somewhat heavier than the Negro youths. (Compare by race "Total—Weighted. . . ." height and weight values, table 2.) Definite differences in both height and weight—the Negro youths being of lower stature and of lighter weight than white—

were noted in studies for former years. (See section: Gains since World War II.)

Inductees versus Youths. Irrespective of age and race, the height and weight data (table 2) show that the inductees were generally taller and heavier than the examined youths. The explanation lies partly in the established minimum height and weight for military service (the minima seemingly outweigh the established maxima), and partly in the fact that the disqualified groups include medically disqualified examinees (in addition to those disqualified for underheight and underweight) whose average height and weight are presumably lower than the total avearge, except for tuberculosis examinees whose height proved to be above the average (Karpinos, 1958b).

FREQUENCY DISTRIBUTIONS BY HEIGHT AND WEIGHT

Height and weight data serve many biologic and socio-economic purposes. To meet the various purposes, frequency distributions by height and weight are as essential as average heights or weights. In the military experience, for example, we may be asked what additional proportion of youth may be expected to be disqualified for military service should the minimum standard height be raised from 60 to 61 inches; or we may be asked to determine what proportion of the military manpower would be available for the selection of men for the military police should the minimum height for such assignment be fixed at 68 inches. In both instances frequency distributions are needed.

Frequency distributions by height and weight were computed for total (white and Negro) youths and inductees, by the following age groups: (17–19), (20–25), and (17–25). The mean height and weight values of these groups and their modal weight values are shown in table 3; the frequency distributions are presented in tables 4 and 5.

The frequency distributions were derived by fitting normal curves to the height and weight data. For height (table 4), the distributions were derived from the numerical values (means and standard deviations) of the variable; for weight (table 5), the logarithmic values (means and standard deviations) of the variable were used. Both procedures were justified by tests of fitness. (See "Appendix: Technical Notes," 3 and 4.)

The frequency distributions are to a base of 100,000. The highest frequencies of these distributions, represented by maximum ordinates,

are shown in the footnotes to tables 4 and 5, for height and weight, respectively. Their corresponding most frequent (modal) values are identical with their mean values—for height, but different from their mean values—for weight, caused by its skewed distributions (table 3: "Modes.")

In addition to the frequency tables, the distributions relating to the (20–25) age group are graphically presented in figures 2-4. The cumulative frequencies of height (fig. 3) are plotted in two ways: the curves starting from the left (bottom) indicate the cumulative frequencies for a particular height or lower; the others starting from the right (bottom) indicate the cumulative frequencies for a particular height or higher.

TABLE 3

Mean heights and weights of youths and inductees and standard deviations of those means; also modes of weight, by race and age groups

(January 1957 through September 1958)

AGE GROUP AND RACE	HEIGHT (INCHES)		WEIGHT (POUNDS)		
	Mean (1)	S.D. (2)	Mean (3)	S.D. (4)	Modes [1] (5)
Youths [2]					
Total (17–25)	68.27	2.82	152.07	26.35	145.85
17–19	67.89	2.79	144.85	25.03	139.15
20–25	68.49	2.83	156.32	26.19	150.26
Inductees					
Total (17–25)	68.91	2.66	157.98	24.33	152.68
17–19	68.39	2.69	149.84	24.35	144.34
20–25	68.96	2.65	158.80	24.16	153.45

[1] Derived from logarithmic normal curve. (See "Appendix: Technical Notes," 4, and footnote to table 5.)
[2] see footnote 3, table 2.

GAINS SINCE WORLD WAR II

General. Like the height and weight data of present-day youths and inductees, those of youths and inductees of World War II were derived from similar data relating to the Selective Service registrants examined for military service (Karpinos 1958b). The military age range in World War II, however, was broader than it is now: it ranged from 18 through 37 years of age, compared with the current age range from 17 through 25 years of age. The comparison between current and World War II data was, therefore, confined to the age groups: (18–19),

and (20–24), and total (18–24). But even when limited to these age groups, a direct comparison between their height and weight values have

TABLE 4

Distribution of male youths and inductees by height, by age groups

(January 1957 through September 1958)

(Base: 100,000) [1]

HEIGHT (INCHES)	MALE YOUTHS, BY AGE GROUPS			INDUCTEES, BY AGE GROUPS		
	(17-19) (1)	(20-25) (2)	Total (17-25) (3)	(17-19) (4)	(20-25) (5)	Total (17-25) (6)
Total	100,000	100,000	100,000	100,000	100,000	100,000
< 57	4	2	2	–	1	–
57–58	15	8	11	6	2	2
58–59	51	29	38	18	6	8
59–60	169	95	117	66	28	30
60–61	436	280	339	208	95	109
61–62	1,067	686	813	568	296	331
62–63	2,265	1.518	1,753	1,409	795	841
63–64	4.220	3,086	3,478	2,880	1,852	1,967
64–65	6,924	5,230	5,750	5,228	3,607	3,790
65–66	9,675	8,008	8,884	8,290	6,455	6,708
66–67	12,623	10,863	11,450	11,480	9,829	9,790
67–68	14,147	13,446	13,381	14,280	12,977	13,117
68–69	13,947	13,892	14,241	14,662	14,856	14,504
69–70	12,095	13,052	12,650	13,480	14,375	14,713
70–71	9,013	11,133	10,491	10,823	12,762	12,320
71–72	6,272	7,924	7,260	7,590	9,558	9,468
72–73	3,716	5,157	4,596	4,649	6,206	6,124
73–74	1,936	2,973	2,628	2,532	3,429	3,371
74–75	887	1,547	1,252	1,136	1,742	1,706
75–76	352	670	559	462	739	710
76–77	131	267	207	164	273	273
77–78	41	96	72	51	87	86
78–79	12	29	21	14	23	24
79–80	2	7	5	3	6	6
> 80	–	2	2	1	1	2

[1] Derived by fitting the data to normal curve, by area method. (See "Appendix: Technical Notes," 3.) The needed values: arithmetic means and standard deviations, were taken from table 3.

Maximum Ordinates: *a*. Youths: Age (17-19)—14,282; age (20-25)—14,077; total: age (17-25)—14,127; *b*. Inductees. Age (17-19)—14,831; age (20-25)—15,071; total: age (17-25)—14,981.

proved misleading, because of differences in the age distributions within these age groups. Consequently, for proper evaluation the values were "standardized."

TABLE 5

Distribution of male youths and inductees by weight, by age groups
(January 1957 through September 1958)
(Base: 100,000) [1]

WEIGHT (POUNDS)	MALE YOUTHS, BY AGE GROUPS			INDUCTEES, BY AGE GROUPS		
	(17-19) (1)	(20-25) (2)	Total (17-25) (3)	(17-19) (4)	(20-25) (5)	Total (17-25) (6)
Total	100,000	100,000	100,000	100,000	100,000	100,000
< 90	232	43	113	78	10	12
90– 95	388	96	196	161	32	35
95–100	805	229	448	399	96	106
100–105	1,512	498	863	824	252	261
105–110	2,542	965	1,526	1,475	523	575
110–115	3,696	1,684	2,448	2,542	1,056	1,128
115–120	5,055	2,542	3,420	3,696	1,784	1,888
120–125	6,380	3,623	4,774	5,055	2,798	2,938
125–130	7,485	4,777	5,703	6,380	4,013	4,179
130–135	8,221	6,154	6,946	7,485	5,301	5,479
135–140	8,511	6,814	7,655	8,221	6,497	6,668
140–145	8,758	7,772	7,984	8,511	7,443	7,937
145–150	8,205	8,054	7,927	8,759	8,403	8,151
150–155	7,355	7,945	7,925	8,206	8,603	8,648
155–160	6,344	7,897	7,247	7,355	8,360	8,351
160–165	5,567	7,180	6,393	6,344	7,758	8,076
165–170	4,486	6,298	5,771	5,567	7,264	6,795
170–175	3,708	5,654	5,044	4,712	6,230	6,411
175–180	2,822	4,663	4,052	3,666	5,435	5,002
180–185	2,222	3,970	3,184	2,785	4,355	4,437
185–190	1,612	3,283	2,603	2,188	3,403	3,244
190–195	1,221	2,500	2,075	1,672	2,890	2,736
195–200	902	1,873	1,523	1,177	2,013	1,894
200–205	615	1,560	1,244	867	1,644	1,535
205–210	441	1,113	769	589	1,156	1,073
210–215	293	789	630	421	804	742
215–220	206	592	467	296	621	570
220–225	143	436	338	191	389	375
225–230	91	314	226	131	296	247
230–235	67	210	161	89	191	182
235–240	40	149	107	56	131	114
240–245	26	105	81	37	89	73
245–250	18	75	52	24	56	52
> 250	31	143	105	41	104	86

[1] Derived by fitting the data to logarithmic normal curve, by area method. (See "Appendix: Technical Notes," 4.) The needed means and standard deviations of weight in logarithms were derived by expressing in logarithms the mid-values of the weight intervals, as obtained from the tabulations, and applying these to their corresponding frequencies. The derived weight values in logarithms were as follows:

a. Youths: Age (17-19)—mean weight 2.15505 pounds and standard deviation .07091 pounds; age (20-25)—mean weight 2.18825 pounds and standard deviation .07031 pounds; total: age (17-25)—mean weight 2.17594 and standard deviation .07235 pounds. These values were computed from the means and standard deviation (in logs) by single years of age, but weighted proportionally by the U. S. male population.

b. Inductees: Age (17-19)—mean weight 2.17013 pounds and standard deviation .06823 pounds; age (20-25)—mean weight 2.19587 pounds and standard deviation .06555; total: age (17-25)—mean weight 2.19363 pounds and standard deviation .06533.

Maximum ordinates (to a base of 100,000): *a.* Youths: age (17-19)—8,668; age (20-25)—8,096; total: age (17-25)—8,098; *b.* Inductees: age (17-19)—8,689; age (20-25)—8,516; total: age (17-25)—8,588.

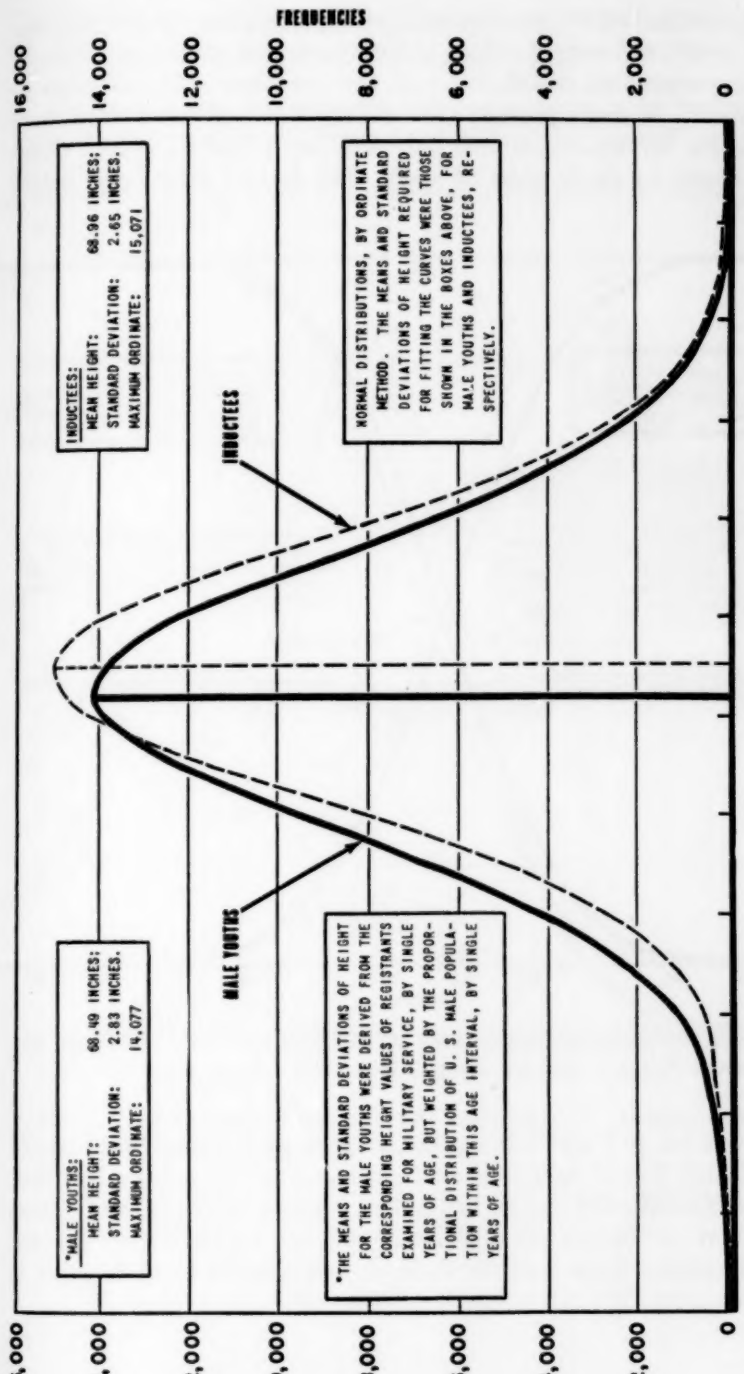

FIG. 2. DISTRIBUTION OF MALE YOUTHS AND INDUCTEES, 20 THROUGH 25 YEARS OF AGE, BY HEIGHT: 1957–1958. (Base: 100,000)

The standardization was accomplished by applying the current age-specific height and weight values of examinees and inductees (table 2) to the corresponding distributions of the examinees and inductees of World War II by single years of age. (While such age distributions were available for the examinees and inductees, no height or weight values were available by single years of age.) The derived height and weight

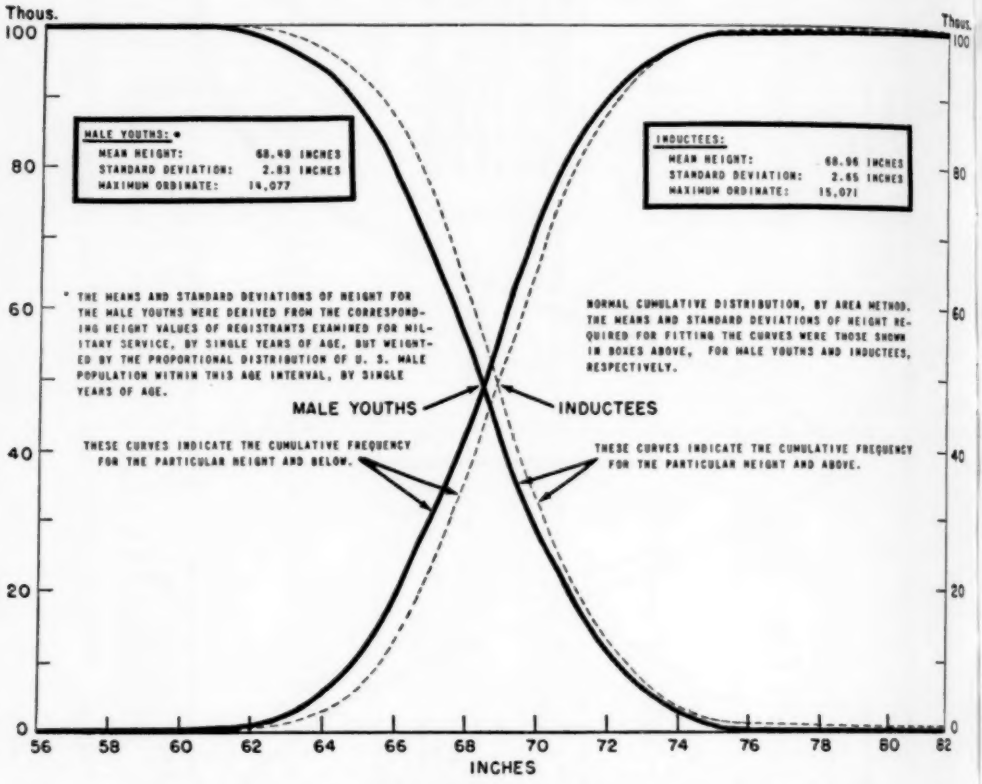
Thous.
100
80
60
40
20
0
MALE YOUTHS:*
MEAN HEIGHT: 68.49 INCHES
STANDARD DEVIATION: 2.83 INCHES
MAXIMUM ORDINATE: 14,077
* THE MEANS AND STANDARD DEVIATIONS OF HEIGHT FOR THE MALE YOUTHS WERE DERIVED FROM THE CORRESPONDING HEIGHT VALUES OF REGISTRANTS EXAMINED FOR MILITARY SERVICE, BY SINGLE YEARS OF AGE, BUT WEIGHTED BY THE PROPORTIONAL DISTRIBUTION OF U. S. MALE POPULATION WITHIN THIS AGE INTERVAL, BY SINGLE YEARS OF AGE.
THESE CURVES INDICATE THE CUMULATIVE FREQUENCY FOR THE PARTICULAR HEIGHT AND BELOW.
INDUCTEES:
MEAN HEIGHT: 68.96 INCHES
STANDARD DEVIATION: 2.65 INCHES
MAXIMUM ORDINATE: 15,071
NORMAL CUMULATIVE DISTRIBUTION, BY AREA METHOD. THE MEANS AND STANDARD DEVIATIONS OF HEIGHT REQUIRED FOR FITTING THE CURVES WERE THOSE SHOWN IN BOXES ABOVE, FOR MALE YOUTHS AND INDUCTEES, RESPECTIVELY.
MALE YOUTHS
INDUCTEES
THESE CURVES INDICATE THE CUMULATIVE FREQUENCY FOR THE PARTICULAR HEIGHT AND ABOVE.
56 58 60 62 64 66 68 70 72 74 76 78 80 82
INCHES

FIG. 3. CUMULATIVE DISTRIBUTION OF MALE YOUTHS AND INDUCTEES, 20 THROUGH 25 YEARS OF AGE, BY HEIGHT: 1957–1958. (Base: 100,000)

values, denoted as "expected" values, show what the height and weight of the World War II examinees or inductees would have been had their age-specific height and weight values been the same as the current values. The "expected" values are contrasted with the actual values; the differences between them indicate changes, not affected by differences in

age (table 6). (This procedure is known as the "indirect" method of standarization. See "Appendix: Technical Notes," 5.)

Youths. When so compared (table 6), namely, by utilizing the "expected" values, the following gains are revealed since World War II.

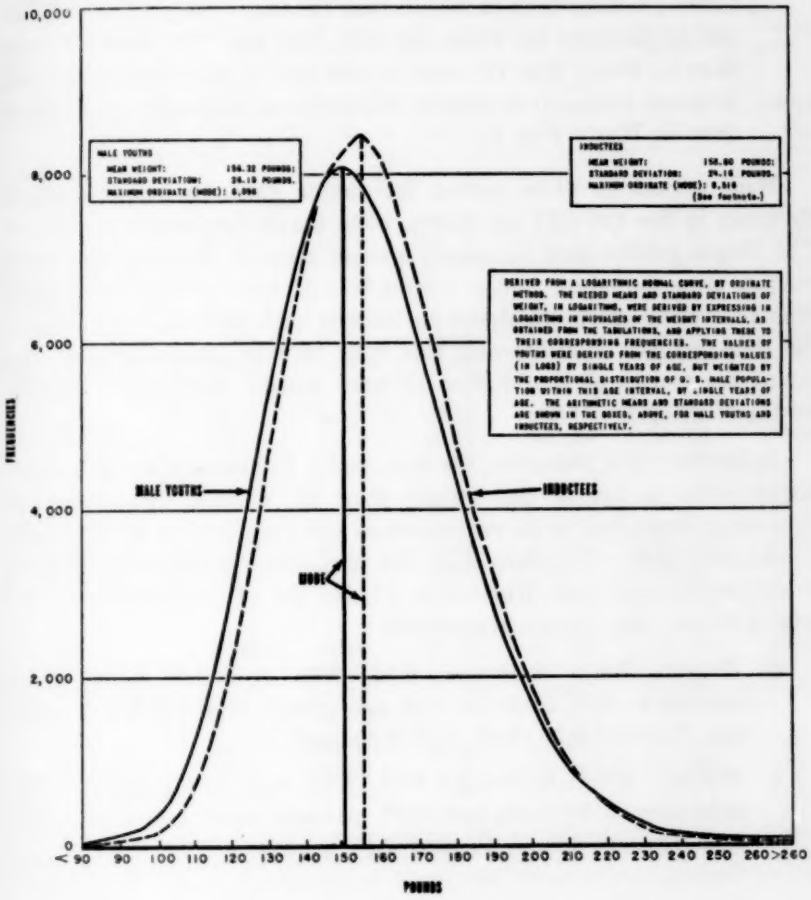

FIG. 4. DISTRIBUTION OF MALE YOUTHS AND INDUCTEES, 20 THROUGH 25 YEARS OF AGE, BY WEIGHT: 1957–1958. (Base: 100,000)

a. *Height*: White youths, (18–19) years of age, 0.08 inch taller; those (20–24) years of age, 0.40 inch taller; and all white youths, (18–24) years of age, 0.23 inch taller, than the white youths of World War II; Negro youths are generally 0.62 inch taller than

in World War II; total youths (white and Negro) are 0.15, 0.44, and 0.29 inches taller within the corresponding age groups than in World War II.

b. Weight: White youths, within the (18–19) and (20–24) age groups and total (18–24) age range, are 4.87, 6.65, and 5.69 pounds heavier than in World War II, respectively; Negro youths within the same age range are 4.82, 5.51, and 5.21 pounds heavier than in World War II; total youths (white and Negro) are 4.86, 6.44 and 5.62 pounds heavier within the corresponding age groups than in World War II.

With respect to white youths, the major gains in height occurred obviously in the (20–24) age group, when maximum height is attained. The Negro youths have seemingly gained more in height, but less in weight, than the white youths within this 15-year period, since World War II. (The gains are shown in table 6 with a "negative" sign, as the data actually indicate—by this type of standardization—to what extent the youths of World War II were shorter and lighter than the youths of today.)

Inductees. For inductees, the data (table 6) indicate for this period larger gains in height and weight than for youths. This could be apparently attributed to the current more strict application of the height-weight standards. The data show for inductees the following gains in height and weight since World War II, for the (18–19), (20–24), and total (18–24) age groups, respectively:

a. Height: White inductees, 0.33, 0.60, and 0.47 inch; Negro inductees: 0.79, inch for each age group; total inductees (white and Negro): 0.38, 0.63, and 0.50 inch.

b. Weight: White inductees: 6.83, 7.61, and 7.24 pounds; Negro inductees: 5.94, 6.39, and 5.97 pounds; total inductees (white and Negro): 6.74, 7.41, and 7.11 pounds.

GAINS SINCE WORLD WAR I

A comparison with World War I data was possible only with respect to inductees, inasmuch as the World War I height and weight data relate to "recruits," namely, individuals examined at the military camps after they had been screened out by the local boards (Davenport and Love, 1921; Karpinos, 1958b). In World War I, local board screening

TABLE 6

Gains in height and weight of male youths and inductees since World War II, by age and race

TYPE OF VALUES AND RACE	MEAN HEIGHT AND WEIGHT, BY AGE: WORLD WAR II					
	MEAN HEIGHT (INCHES)			MEAN WEIGHT (POUNDS)		
	Total (18-24) (1)	(18-19) (2)	(20-24) (3)	Total (18-24) (4)	(18-19) (5)	(20-24) (6)
Youths [1]						
White						
Actual	68.12	68.06	68.19	146.29	143.70	149.30
Expected	68.35	68.14	69.59	151.98	148.57	155.95
Difference	—.23	—.08	—.40	—5.69	—4.87	—6.65
Negro						
Actual	67.79	67.64	67.91	145.24	141.85	147.85
Expected	68.41	68.26	68.53	150.45	146.67	153.36
Difference	—.62	—.62	—.62	—5.21	—4.82	—5.51
Total						
Actual	68.07	68.01	68.14	146.13	143.47	149.04
Expected	68.36	68.16	68.58	151.75	148.33	155.48
Difference	—.29	—.15	—.44	—5.62	—4.86	—6.44
Inductees [2]						
White						
Actual	68.20	68.17	68.24	147.14	144.82	149.79
Expected	68.67	68.50	68.84	154.38	151.65	157.40
Difference	—.47	—.33	—.60	—7.24	—6.83	—7.61
Negro						
Actual	67.85	67.68	67.97	145.80	142.36	147.88
Expected	68.64	68.47	68.76	151.77	148.30	154.27
Difference	—.79	—.79	—.79	—5.97	—5.94	—6.39
Total						
Actual	68.16	68.12	68.20	146.95	144.56	149.51
Expected	68.66	68.50	68.83	154.06	151.30	156.92
Difference	—.50	—.38	—.63	—7.11	—6.74	—7.41

[1] "Actual" values taken from Karpinos, 1958b; "Expected" values derived by applying the current age-specific height and weight values of examinees (table 2, "Examined") to the distribution of World War II examined registrants by single years of age.

[2] "Acutal" values taken from Karpinos (unpublished data): "Expected" values derived by applying the current age-specific height and weight values of inductees (table 2, "Inducted") to the distribution of World War II inductees by single years of age.

was of primary nature and was not restricted to eliminating men with manifestly disqualifying defects, as it is the case now and as it was in World War II. The height and weight data of World War I recruits closely parallel thus the current and those of World War II inductees.

In comparing World Wars I and II height and weight data, it was found that the recruit of World War II was about two-thirds (.67) of an inch taller and 10.7 pounds heavier than the recruit of World War I (Karpinos, 1958b).

Thus, the recruit of today is on the average 0.5 inch taller and somewhat more than 7 pounds heavier than the recruit of World War II, and about 1.2 inches taller and 18 pounds heavier than the recruit of World War I.

In part, the gain in weight is to be attributed to the gain in height. Current weight-height standards provide for an average increase of 3 to 4 pounds per inch of height (Karpinos, 1958a, and Metropolitan Life Insurance Company, 1960). Seemingly, there has been a net gain of around 14 pounds in the average weight of the recruit, in the (18–24) age group, since World War I.

SUMMARY

1. The study is based on height-weight data abstracted from the medical examination reports of youths examined for military service during the 21-month period, from January 1957 through September 1958.

2. It was a sample (50%) study that contained some 273,000 medical examination reports.

3. The age range of these examinees was 17 through 25 years. Their mean age was around 22 years.

4. The analysis was carried out by race (white; Negro) and by single years of age within each race—separately for inductees and all examinees.

5. The mean heights and weights for all youths within the (17–25) age range were computed as follows, by race: white youths—68.3 inches (173.5 cm) and 152.3 pounds (69.1 kg); Negro youths—68.4 inches (173.7 cm) and 150.7 pounds (68.4 kg); total (white and Negro) youths —68.3 inches (173.5 cm) and 152.1 pounds (69.0 kg) (tables 2 and 3).

6. Due to certain selective factors, the inductees proved to be taller and heavier than the examined youths. Their average heights and weights, by race, were computed as: white inductees—68.9 inches (175.0 cm) and 158.4 pounds (71.85 kg); Negro inductees—68.8 inches (174.8

cm) and 155.0 pounds (70.3 kg); and total (white and Negro) inductees —68.9 inches (175.0 cm) and 158.0 pounds (71.7 kg) (tables 2 and 3).

7. For both white and Negro youths, maximum heights are indicated for age 22: 68.9 inches (175.0 cm) for white youths, and 68.7 inches 174.5 cm) for Negro youths (table 2). Maximum weights are 159.4 pounds (72.3 kg) for white youths, age 22, and 156.2 pounds (70.9 kg) for Negro youths, age 23.

8. In addition to mean height and weight values, separate frequency distributions of youths and inductees by height and weight were derived, for certain age groups (tables 4 and 5; figures 2-4).

9. Comparison with height and weight data of World Wars I and II indicates that the recruit of today is about 1.2 inches (3.05 cm) taller and 18 pounds (8.2 kg) heavier than the recruit of World War I, and about one-half inch (1.27 cm) taller and somewhat more than 7 pounds (3.2 kg) heavier than the recruit of World War II.

APPENDIX: TECHNICAL NOTES

1. *Adjustments for Disproportionate Representation of the Subclasses.* While the Army regulations require that a copy of the medical report of each disqualified examinee be forwarded to the Surgeon General's Office, Army, there is no similar requirement with respect to qualified registrants. Copies of the reports of qualified registrants are received after these registrants have been inducted. But a certain number of the qualified registrants enlist prior to induction, others may not be called at all. Besides the number of qualified registrants forwarded for induction at any particular time is determined by the "calls" for induction, as issued by the Department of Defense. Since height and weight values of the inductees, taken as representative of the qualified registrants, proved to be different from those of disqualified registrants, it was obviously necessary for proper evaluation of total examinees to ascertain first the proper ratios of these subclasses. (Had there been no such differences, no adjustments would have been necessary.) The ratios of qualified to disqualified for this period were determined as 7:3, for white examinees, and 5:5, for Negro examinees—clearly different from the ratios of inducted and disqualified indicated by table 1. (The ratios used here differ somewhat from those used in previous studies, Karpinos, 1960, as further adjustments were made in the present study for enlistments and local board disqualifications.) The current data were "weighted" in accord with these ratios.

2. *Height and Weight Values of Youths.* In combining the age-specific values of youths ("examined") by age groups (table 3) further adjustments were necessary because the distribution of all U. S. male youths by single years of age within the military age range is very different from the age distribution of youths examined (fig. 1). Consequently, in obtaining height and weight values of all youths by age groups and for the total age range, the age-specific values were age-adjusted ("weighted") according to the proportional distributions by age of all male youths in the U. S. population. The values for "total" (white and Negro) were computed by additional "weighting" the separate white and Negro height and weight values by the racial distribution of youth within each single year of age, according to the Census distribution.

3. *Fitting of Normal Curve to Height.* The suitability of fitting a normal curve to the arithmetic values (means and standard deviations) of height was established by higher moments, namely, by computing β_1 and β_2 (Elderton, 1953; Smith and Duncan, 1945). The actual fitting of the data was accomplished by the usual procedures (See Croxton and Cowden, 1955, pp. 594-603).

4. *Fitting of Normal Logarithmic Curve to Weight.* Because of the characteristic skewness of the weight distributions, the suitability of fitting a normal logarithmic curve to the weight data was tested by the formula given by Davies, 1929. The logarithmic coefficients of skewness calculated from the data by this formula were by far less than 0.15, given as the criterion for logarithmic normality. (See Davies and Crowder, 1933, pp. 303-306; also Croxton and Cowden, 1955, p. 619.)

The procedures outlined by Davies (1929; table IV) were followed in deriving the frequency distributions (table 5 and figure 3), as well as the modal values (table 3).

5. *Expected Values.* The expected ("standardized") values given in table 6 were computed by what is known as the "indirect" method of standardization. It was used here because the height and weight values for World War II were not available by single years of age. The standardization consisted of applying the current age-specific height and weight to the age distribution of the World War II population (examinees and inductees) which was available by single years of age. The values obtained in this manner are referred to as "expected" values. They show what would have been the height and weight of World War II values in accordance with the current values. The differences between

the actual and "expected" values reflect thus gains (or losses) due to changes in these values. (See Stouffer, 1931, for a detailed discussion of this method of standardization.)

ACKNOWLEDGMENTS

I am much indebted to the many persons in the Medical Statistics Division, Office of The Surgeon General, Department of the Army, who were involved in this study; specifically, to Betty Feldman, Lillian Smith, Josephine Balthazar, and their coworkers, who carried the brunt of the numerous required computations. Special acknowledgments are made to Marguerite Wood for accomplishing the more complex computations, and Eurselene Martin for her most faithful checking (including certain computations) and typing of the manuscript.

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THE URINARY EXCRETION OF β-AMINO-ISOBUTYRIC ACID IN PACIFIC POPULATIONS

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THE differential excretion of β-amino-isobutyric acid is under genetic control. Family studies are consistent with the hypothesis that a single major gene pair is responsible for most of the variation, although other genes and environment may contribute to the variation. High excretors are homozygous for a single recessive gene and low excretors either heterozygous or homozygous for the dominant allele (Harris, 1953; Gartler, Firschein, and Kraus, 1957). The frequency of these genes varies considerably in different populations (table 1, figure 1).

The excretion of BAIB may be markedly increased under conditions of excessive tissue destruction; *e.g.*, leukemia, tuberculosis, starvation, following nitrogen mustard therapy, and after exposure to radiation. In 1959 Blumberg and Gartler studied the urinary excretion of BAIB in 188 inhabitants of Rongelap Atoll, Marshall Islands, U. S. Trust Territory of the Pacific Islands (Blumberg and Gartler, 1959). An extremely high frequency of high excretors (86%) was found in this population. Sixty-five of these individuals had been accidentally exposed to fallout in March, 1954, following the detonation of a high-yield thermonuclear device during experiments at Bikini Atoll in the Pacific Proving Grounds (Conard, *et al.*, 1960). The frequency of high excretors was no higher in this group than in the 119 unexposed and 4 individuals whose exposure history was not known who had lived on Rongelap with the others since their return to the island in 1958. Furthermore, there was no correlation between the levels of the body burdens of either caesium 137 or zinc 65, and the BAIB excretion, in both the exposed individuals and the controls. The levels of these radioisotopes was approximately equal in both of the groups who had been living side by side on the island since their return. Since there was no direct evidence that the radiation was the cause of the high frequency of high BAIB excretion, several alternate explanations were suggested. One of these was that there might be an Asian focus for high frequencies of the high BAIB excretor

TABLE 1

Frequency of high excretors in various populations

POPULATION	NUMBER STUDIED	PERCENT HIGH EXCRETORS	REFERENCE
Americas			
Apache Indians, Arizona	110	59	Gartler, Firschein and Kraus, 1957
Pima Indians, Arizona and Northern Mexico	118	50	Gartler and Kraus, quoted in Gartler, 1960
Papago Indians, Arizona	42	50	" "
Athabascan Indians, Alaska	25	56	Allison, Blumberg and Gartler, 1959
Carib Indians, Galibi, Surinam	39	23	Blumberg and Gartler, present study
Eskimos, Alaska	120	23	Allison, Blumberg and Gartler, 1959
Black Caribs, New York and British Honduras	247	39	Gartler, Firschein and Kraus, 1957
Negroes, New York	40	29	Gartler and Firschein, quoted in Gartler, 1960
U. S. Whites	218	10	Gartler, 1960
Europe			
British White	345	10	Harris, 1953
Italian Whites	792	7	Colchi-Novati *et al.*, 1953
Africa			
East Africans (Tanganyika)	40	40	Gartler and Allison, unpublished
Asia			
Chinese	33	45	Sutton, 1960
Japanese	41	42	" "
South Asia and Oceania			
Vietnamese	187	26	Blumberg and Gartler, present study
Javanese, Surinam	140	22	" " "
New Guinea Highlanders	70	9	" " "
Micronesians			
Rongelap, M. I.	188	86	Blumberg and Gartler, 1959
Utirik, M. I.	18	83	" " "
Ebeye Islet,Kwajalein, M. I.	34	[illegible]	Blumberg and Gartler, present study
Polynesians, Tahiti	46	12	" " "

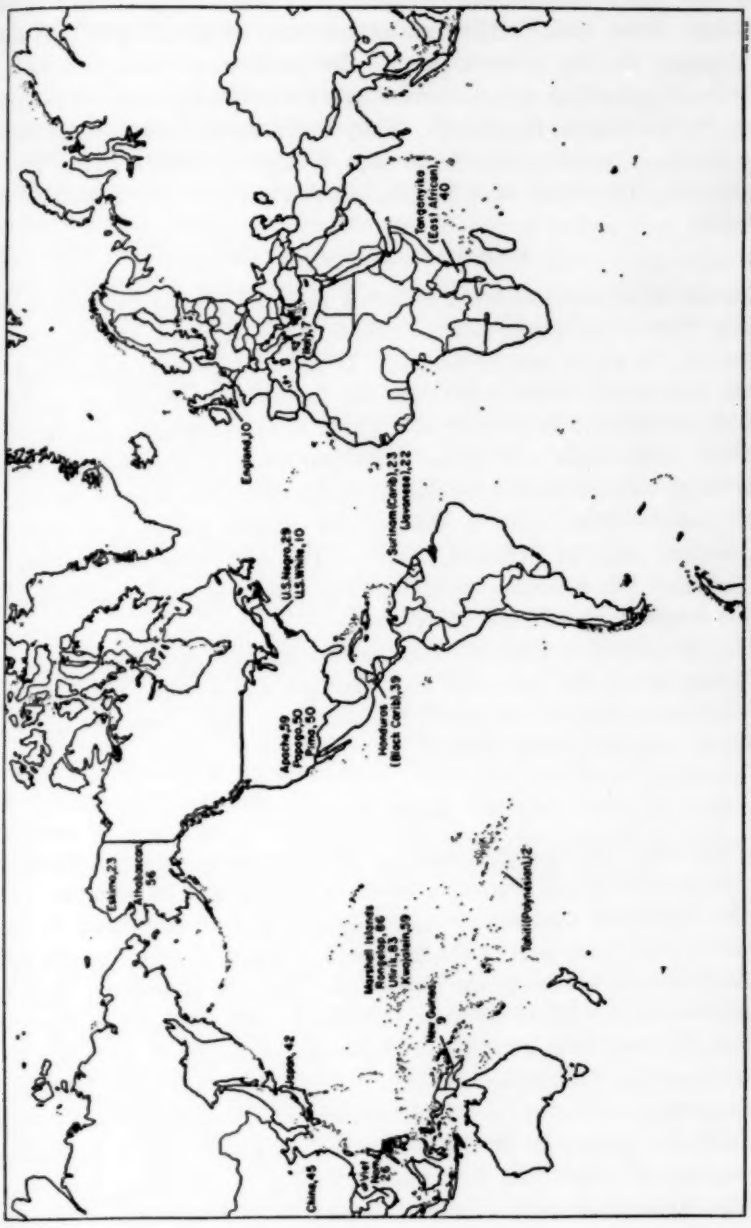
THE PREVALENCE IN PERCENT OF HIGH EXCRETORS OF BETA-AMINO-ISOBUTYRIC ACID IN VARIOUS POPULATIONS.
England,10
Tanganyika (East Africa), 40
Surinam(Carib),23
Leeuwards),22
U.S.Negro,29
U.S.White,10
Apache,59
Papago,50
Pima,50
Honduras (Black Carib),39
Calmo,23
Chinook 56
Marshall Islands Rongelap, 66 Utirik,83 Kwajalein,59
Tahiti(Polynesian),12
New Guinea
Hopi 42
China,45
Pueblo Indian 28

FIG. 1.

gene. To test this hypothesis, collections of urine samples from several Southeast Asian and Pacific populations have been initiated and some are reported in the present paper. In most cases sample sizes and methods of collection are not satisfactory for establishing an accurate figure for phenotype frequency. They are suitable, however, to determine if an extremely high frequency of high excretors exists in the population. The study of a South American Indian population is also included.

MATERIALS AND METHODS

Casual urine samples were collected in small plastic containers containing thymol as preservative. These were shipped in refrigerated containers, by air or sea, to Seattle. If the studies were not completed within 2–3 weeks of the collection, the urines were stored at —20° C. until the tests could be performed. There is no appreciable deterioration of BAIB under these conditions. Determinations of BAIB were carried out by high voltage electrophoresis on paper using the ratio of the optical density of the BAIB spot to that of the neutral amino acid spot as a comparative measure (Gartler, 1959). The anti-mode in the distributions studied was between .30-.35 and this class was used as the dividing point between high and low excretors.

The frequency of high excretors is independent of sex and age, with the exception of the five year and younger age group, where there is some evidence for an increased frequency of high excretors (Gartler, Firschein and Gidaspow, 1956).

POPULATIONS

These are indicated in table 1. The Javanese were inhabitants of the village of Moengo, Surinam, South America, and its suburbs. They are the immediate descendants of Javanese who had emigrated to South America in the early part of the twentieth century. There were 66 males, 76 females and two subjects whose sex was not recorded. The ages range from 3–73. There were six children 5 years or under in the group. Individuals who had known non-Javanese antecedents were excluded. The samples were assembled casually during three collection trips to the Javanese area. The Caribe Indian specimens were collected in the village of Galibi at the mouth of the Marowyne (Maroni) River in Surinam. There were 22 males and 18 females ranging in age from 6–50.

The Polynesian samples were collected from Polynesian members of a military unit on the island of Tahiti. Since there is universal obligated

male military service, this collection approximates a random sampling of males from the ages of approximately 18–30 years.

The New Guinea samples were from Eastern Highland people of the Australian Trust Territory of New Guinea with the exception of two specimens which were from Western Highland people. The sample included individuals with and without Kuru and the population is described elsewhere (Gajdusek and Zigas, 1961). The specimens were selected casually from the collections made during a survey of the Highland people by Dr. D. C. Gajdusek and his co-workers. The sexes were equally distributed and none of the subjects was younger than 5 years. Since there was no difference in the frequency of high excretors in Kuru and non-Kuru individuals, they were combined.

The Vietnamese samples were collected during the course of the nutrition survey of that country conducted by the Interdepartmental Committee on Nutrition for National Defense (ICNND). Random collections in various communities were made by methods described elsewhere (Republic of Vietnam, 1960). The urine samples made available to us were selected casually from these. The ages ranged from 4 to 69 years, and two subjects were 5 years or less. Of the 185 samples, 150 were from individuals classified as South Vietnamese. The remainder included 12 individuals from an orphanage where South and North Vietnamese and Chinese children were housed, 15 North Vietnamese and 8 individuals classified as "mountaineers." There were no significant differences in the several groups and they were combined.

Thirty-four urines were collected from Micronesians living on the islet of Ebeye in Kwajalein Atoll, Marshall Islands. Many of these were employees of the United States Government on this atoll, and they originated from other atolls in the Marshall Islands group.

RESULTS

The prevalence of high excretors in the populations described above, compared to other populations on which published data is available, is shown in table 1 and figure 1. The frequency distribution of urinary excretion is shown for the Javanese and Vietnamese in figure 2.

The prevalence in Rongelap, Utirik and Ebeye, taken separately or combined, is significantly higher than any of the other Pacific and circum-Pacific populations tested. The total Marshall Islands sample of 240 individuals represents approximately 2% of the Micronesian population of the Marshall Islands.

The sample size and method of collection preclude the use of some

of these samples as accurate estimates of the prevalence of the high excretor phenotype in these populations. They are sufficient, however, to indicate that it is improbable that the high excretor rate is as high in any of the populations surveyed as it is in the Micronesians. Our

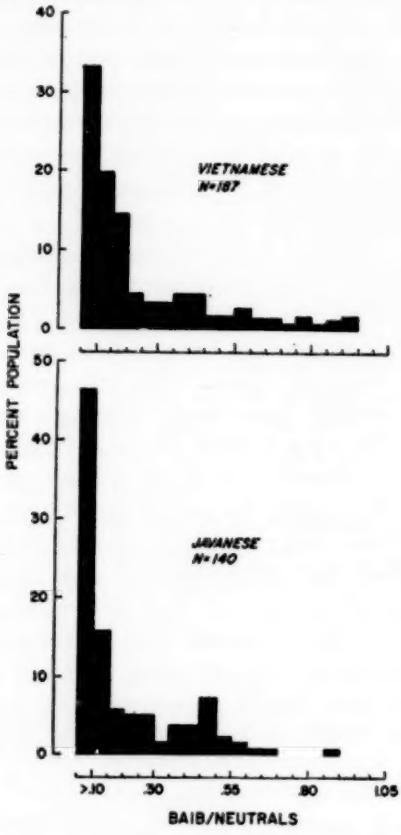

FIG. 2. THE FREQUENCY DISTRIBUTION OF URINARY EXCRETION OF B.A.I.B. IN VIETNAMESE AND JAVANESE.

findings, therefore, are inconsistent with the explanation that the high prevalence in Micronesia is due to a high Asian focus for the high-excretor gene. Further studies on Asian populations will be needed, however, before this possibility can be ruled out. Other tenable explanations for the high frequency include: 1) the possibility of the operation

of random genetic drift in these island populations and 2) that some environmental factor has operated to increase the frequency of high excretors in the Marshall Islands. Limited family studies on Rongelap were consistent with the genetic hypothesis (Blumberg and Gartler, 1959).

CONCLUSION

The high prevalence of high excretors of BAIB found in the Micronesians from the Marshall Islands is not reflected in four other Pacific and Southeast Asian populations studied. Further investigations are suggested. The prevalence of high excretors in other populations of the world are reviewed.

ACKNOWLEDGMENTS

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BOOK REVIEWS

Human Races. By S. M. Garn. 137 pp. C. C. Thomas, Springfield, Ill., 1961. $5.50.

This is a felicitous attempt to describe what constitutes race and the mechanisms of racial differentiation. The author stresses the dynamic features as opposed to the more traditional and deeply entrenched static concepts, and refers to the development of races as "human raciation," implying a process rather than a condition. He also takes pains to dispel the lingering illusion that there are some mystical, almost divine forces at work which create and maintain a "proper" racial differentiation of men. "A race in man," the author reminds us (p. 6), "is a population, a population of men, women and children, of fathers, mothers, and grandparents." We are urged to recognize that even in the absence of admixture human raciation continues as an on-going process; that races do change their genetic makeup continually and that it is no longer sufficient to merely note the existence of races and describe their differences. We must center attention on the mechanisms that bring about the changes. Since new race populations are forming all the time, the study of race formation is an important and fascinating topic. This point of view comes at a time when it is considered by some all but unrespectable to speak of races and thereby admit the existence of racial differences among men.

In the last century the enumeration of the races of men has run from but two to fifty or more for Europe alone. The author interprets this tendency in race taxonomy to either "lump" or "split" as a result of an overly elastic use of the term race. The adoption of standard, agreed-upon taxonomic units of reference would greatly facilitate communication between students of race. Eschewing the setting up of a completely new terminology, the author suggests that the terms *Geographical Race* and *Local Race*, introduced by Rensch in 1929, be employed because they explicitly state which taxonomic unit is involved. Geographical races are broad, geographically-delimited population collections which probably do not exceed ten in number. An example of this degree of taxonomic magnitude is the Amerindian Geographical Race.

Local Races are units isolated by distance, geographical barriers or social prohibitions and tend to be totally or largely endogamous with only very small amounts of gene-flow from contiguous and related Local

Races. The Navajo, Hopi, or Zuni are examples of Local Races in the Amerindian Geographical Race system. Local Races, it is pointed out, probably run into the hundreds.

Drawing upon the term microgeographical race introduced by Dobzhansky, Garn finds the term *Micro-Race* most applicable in densely populated areas of Europe and Asia where each town and hamlet may be genetically somewhat distinct from the others. Some of the differences at this level may be so slight as to be detectable only by the trained observer or the serologist. Micro-Races obviously number in the thousands.

With regard to the race differences which make taxonomic schemes possible, we are not bound exclusively to phenotypically expressed factors. Such hereditary traits as blood groups, the haptoglobins, taste blindness, fingerprints and rare hereditary diseases are becoming increasingly useful in analyzing ongoing evolution within races. Not only can we know the frequencies of such traits, but since their mode of inheritance is known, it is also possible to determine gene frequencies so that some limited racial distinctions on the basis of genotype can be made. Garn holds out hope that other racially significant polygenic traits of as yet unknown mode of inheritance will be genetically worked out and appropriate mathematical methods of handling the data will be developed so that an increasing amount of racial taxonomy will rest on genotype.

In the chapter dealing with natural selection and race, the author spells out the increasingly well-recognized relationship between race and environment. The heat, light and ultra-violet portions of the solar spectrum are stressed as major "race makers" and numerous specific human adaptations to special environmental conditions are illustrated.

Many readers will be particularly interested in the relationships between race and culture. It is pointed out that the influence of culture or learned behavior on maintaining genetic isolates we call races may be very great. The role of culture in disease-race relationships is illustrated by reference to how man has made much of Africa malarial through slash-and-burn agriculture and thus has caused an increase in the appearance of the sickle-cell trait in its population. Additional thought-provoking material in the same chapter deals with the possible adaptive nature of hereditary diseases, such as coronary illness and diabetes.

Some important statements and suggestions are to be found in the chapter dealing with race behavior and intelligence. As unpopular as such suggestions may be in some quarters these days, Garn asserts that it would be a mistake to ignore intelligence completely in considering race differences. In the author's opinion, human races are probably

comparable in the sum total of what we call "intelligence" but differ in details. Some of these details may involve such functions as form discrimination, color sense, tonal memory, mechanical reasoning, abstract reasoning, and other aspects of intelligence. Some of these skills, it is suggested, may have had different adaptive value in different areas.

Human Races is a book which can be highly recommended to professionals and students alike. The professional might wish, however, that its 132 pages of text had been expanded to include an elaboration of various materials only tantalizingly touched upon. In spite of its brevity (and perhaps, to some extent, because of it) it stands out as one of the best of the attempts to elucidate the "what," "why," and "how" of race.

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Medizinische Genetik: Eine Einführung in ihre Grundlagen und Probleme. By W. LENZ. Stuttgart, Georg Thieme Verlag, 1961. viii + 197 pp. 66 figures and 35 tables. $5.50.

This is a modern, comprehensive treatise of human genetics. As genetics ascertains the existence of its subject matter by default (that is by studying cases in which the hereditary factors are missing or replaced by their mutant counterparts), any book on human genetics must inevitably deal with hereditary abnormalities and pathological conditions. Thus the title, medical genetics, is quite appropriate.

Although most of our knowledge of the machinery of heredity comes from studies of plants and invertebrate animals, in one respect human —or medical—genetics has been far ahead of the genetics of other organisms. As Beadle pointed out in his classical review in 1935, chemical genetics, concerned with the basic problem of the mode of action of hereditary factors, started with studies on human beings. The remarkable pioneering discoveries of A. E. Garrod of the "inborn errors of metabolism," made almost simultaneously with the re-discovery of Mendel's laws, constituted a fruitful model for the physiological-biochemical approach to problems of heredity. Unfortunately, professional geneticists hardly knew about Garrod's work or greatly misjudged its significance. Yet, it was the first clear indication that genes act through specific and definite biochemical reactions. Today, after the spectacular success

of microbial chemical genetics, the significance of the work of the pioneer human geneticists is again overshadowed, but diseases like sickle cell anemia and other hemoglobin anomalies are still providing new leads. This makes human genetics one of the most promising and most vigorously growing branches of biology.

The book under review confirms this view. As its author, a reader ("Privatdozent") at the Pediatric Clinic of Hamburg University points out in the Preface, the recent advances in human genetics consist not merely of an accumulation of further data but rather the very foundations of the subject are in the process of being rebuilt or even newly constructed.

The book is divided into five parts: (1) General introduction, (2) The nature of the genes, (3) Simple gene effects, (4) Mutations, and (5) Complex gene effects. At the end of individual chapters (in some cases even after each sub-division of a chapter) short, useful lists of references are given. There is no general author index. There are, however, two highly useful glossaries, one for the genetic, the other for the medical terminology. As many of the readers will not be equally versed in both genetics and medicine, they will be grateful for the one or the other.

The book is well written, well illustrated, and quite up-to-date. Of the five parts perhaps the fifth (Complex gene effects) is the most valuable for those who wish to penetrate the foundations of modern genetics in general and of human genetics in particular. Here the author fully realizes his intention to give a clear exposition of the methodology and the way of thinking ("Denkweise") in human genetics, rather than to present a comprehensive list of facts and figures. Phenomena, like the concerted effect of non-allelic genes, variability in gene manifestation (penetrance, expressivity), phenocopies, polygeny and others, which so often give geneticists headaches, are described and explained with great lucidity. In dealing with hybrid vigor ("Luxurieren der Bastarde," pages 163 ff.) the author fails to refer to modern views about co-adapted gene complexes or super-genes (Mather, Wallace, Dobzhansky). Highly praiseworthy is the treatment of human chromosome aberrations, a recently discovered phenomenon. In this field the author himself has made significant contributions.

The book is a readable, useful and reliable introduction to human genetics as it is today.

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Onsets, Completions, and Spans of the Osseous Stage of Development in Representative Bone Growth Centers of the Extremities. By S. I. PYLE, H. C. STUART, J. CORNONI, and R. B. REED. 126 pp. Child Development Publications, Purdue University, Lafayette, Indiana. 1961. $3.25.

With its 10 tables and 46 figures, this report from the Harvard Longitudinal Studies of Child Health and Development constitutes an interim summary of radiographic research on the appearance and completion of 21 selected centers of ossification, examined in a total of 78 boys and girls. Here, the term "onset" refers to interpolated data on the age-at-appearance of centers in which "the major consideration in assigning onset age by interpolation is the developmental status of the nodule when it is first seen." Completion, in turn, "denotes the age at which the process of ossification first appears to have ended." As with onset, interpolation apparently has been the rule for completion, necessitated in large part by one-year class intervals after the sixth year of age.

The 21 centers of ossification included in the present study appear to have been selected on the basis of representativeness, including a variety of round bones and epiphyses of tubular bones though the exact reasons for selecting individual centers are not totally clear.

Many of the findings in this study are straightforward, as for example, the means and standard deviations for time of onset, completion and "span" for the 21 centers considered in 34 boys and 44 girls which agree well with Pyle and Sontag (Amer. J. Roentgenol., 1943). While median values and percentiles would perhaps have been more appropriate for these ossification data, the small sample sizes involved make it impossible to demonstrate major skewing in any event. Other findings in this particular study support the evidence that the time of appearance of individual centers of ossification is only slightly related to the age at completion of the same centers. The present findings, furthermore, document negative correlations between the age at onset and "span." As other investigators have demonstrated, the later the age at onset, the shorter the span to completion, possibly indicating a more steeply ascending curve of steroid hormone secretion in the late-ossifying children.

There are some problems inherent in this report of investigations on children born in the early 1930's and effectively completed a decade ago. There is, of course, sample selection and its description and the presumed

exclusion of the children whose growth patterns were least satisfactory. More pertinent, however, is the obviously attenuated sample of boys. The reason for the much smaller number of boys (34) than of girls (44) was the still-incomplete maturational status of some of the boys at age 18, the last age at which radiographs were taken. Under these circumstances, the age-at-completion data on the boys in table 1 are drawn from an attenuated distribution and are therefore somewhat below the true values that might obtain in the population sample as a whole.

Included among the many tables are raw data on age-at-onset and age-at-completion for individual bones in individual children, a feature that makes the raw data available to other workers. The many figures further show the age at onset and age at completion and therefore the "span" for individual centers plotted on a per-child basis, which are compared to the standard deviations of the centers in question. It is thus apparent how extraordinarily great individual variability in osseous timing, span and completion may be and how low the correlations are between one bone and another. Under these circumstances, it is more than apparent that the concept of overall "bone age" is a rather nebulous one, and the tool—though clinically useful—is hardly in accordance with the original assumptions concerning bone ossification.

This report fully documents individuality in bony behavior though without particular reference to the source of the individual differences. No attempt is made to exploit the twin or sibling data of the Harvard Longitudinal Studies of Child Health and Development, nor are genetic and non-genetic sources of variability apportioned. Thus it is that the present investigation, the culmination of the 30-year longitudinal program, is most useful in showing variability. It leaves to other investigators the problem of investigating the sources of inter-child and intra-child variance in ossification and osseous union. For that matter, this study also leaves open the validity of the exact measures chosen, the question of exactly when a round bone is "completed" and when an epiphysis is fully united to its diaphysis.

The 12-item bibliography is not intended to be exhaustive, including 5 items published before 1950 and 2 recent articles still not available. The study, therefore, constitutes a meticulous analysis and descriptive documentation of certain aspects of ossification in a selected number of bony centers, from two rather small subsamples of Boston children.

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Evolution after Darwin. Edited by Sol Tax. Vol. 1, vi + 629 pp. Vol. 2, vi + 473 pp. Vol. 3, viii + 310 pp. University of Chicago Press. Chicago, Illinois, 1960. $25.00.

In November 1959 the University of Chicago celebrated the centenary of Darwin's *Origin of Species,* published on 24 November 1859 in an edition of 1,250 copies (*not,* as the editor says in his Preface, in an edition of 1,500). For five days some fifty scientists from ten nations were brought together to present their papers for a series of panel discussions. Sol Tax, Professor of Anthropology at the University of Chicago, was Chairman of the Centennial Celebration and together with various colleagues was responsible for the selection of the scientists invited to be the stellar performers.

The Evolution of Life. $10.00.

Volume I is devoted to *The Evolution of Life: Its Origin, History and Future.* Most appropriately the first contribution is from the pen of Sir Julian Huxley, "The Emergence of Darwinism." This is one of the best short accounts of the origin of Darwin's thought available. I am a little disappointed to find that Huxley does not do quite the justice they deserve to Darwin's views relating to the effects of cultural factors upon the evolution of man. It is in *The Descent of Man* (1871) that Darwin deals most fully with this aspect of man's evolution, and he does so in the first 300 pages of a book of more than 1,000 pages. The 700 which are largely devoted to presenting the case for sexual selection apparently serve to blot out the memory of what Darwin wrote in those considerably more important preceding pages. Thus, Darwin is no exception to the rule, that the lesser part of their work often lives on while the better is interred with their bones. This certainly seems to have been the case in respect of *The Descent of Man.*

Huxley, with his customary felicity for coining the telling phrase, points out that in its broadest terms "the biological phase of evolution stems from the new invention of self-reproducing matter; the human phase, from that of self-reproducing mind," and that it is by the development of this second mechanism that man has established his unique position in the world. In the psychosocial phase evolution is primarily cultural, and within the framework of human societies is subject to psychosocial selection. Natural selection operates blindly; psychosocial selection operates purposefully, it is a goal-selecting mechanism, and that

goal is greater fulfillment—"the fuller realization of more possibilities by the human species collectively and more of its component members individually." "We shall need a science of human possibilities to help guide the long course of psycho-social evolution that lies ahead." This is an admirable suggestion, and it is to be hoped that it will bear fruit.

Harlow Shapley ("On the Evidences of Inorganic Evolution") conducts us on an intergalactic tour which is designed to put man's egocentrism in its proper place—a refreshing and informative experience. A convincing case is made for the existence of life in systems other than our own planetary system in this universe. Hans Gaffron deals with the complex question of "The Origin of Life." This, again, is one of the clearest and most cogent discussions of this subject I have read. E. A. Evans, Jr., puts the viruses in proper perspective in relation to the continuum of life. Bernhard Rensch in "The Laws of Evolution" has produced a most useful, concise conspectus of those laws with which all students of organic evolution should be familiar. In a long (pp. 117-180) and valuable essay George Gaylord Simpson discusses the history of life from the paleontological viewpoint, and concludes that the history of life on this earth has involved, in the main, four grand processes: expansion, progression, equilibrium (or stabilization), and extinction. "Nothing in the recorded history of life arises *de novo*. All is transformed from what went before" is a principle to remember. In his usual original manner, Simpson considers some philosophical problems: the problems of order, utility, progress, and purpose. E. B. Ford ("Evolution in Progress") effectively demonstrates the evolution of heterozygous advantage in polymorphism. He recalls how this fact makes it possible to predict certain evolutionary relationships which are not obvious upon mere inspection. It was, indeed, Ford who recognized in 1945 that since the human blood groups were polymorphic they must be balanced in the population by contending advantages and disadvantages. From this Ford predicted that members of different blood groups would not be equally susceptible to specific diseases—a prediction which has been abundantly supported. It is a fact worth noting that the blood groups were at first thought to be particularly valuable in tracing the relationships between the varieties of man because they were considered to be adaptively neutral. The recognition that their frequencies are selectively adaptive places the blood groups in their proper setting, as adjustments to the genetic constitution of each breeding population, changes within which will be reflected in the blood group frequencies. And as Ford points out, it is the concept of polymorphism that invests the blood groups with their significance for human evolution.

In "The Comparative Evolution of Genetic Systems" G. Ledyard Stebbins shows how closely genetic systems are correlated with the complexities of the organisms involved, and makes the point that in the higher animals and plants the commonest genetic system is one that promotes a maximum of evolutionary flexibility by genetic recombination. Daniel I. Axelrod, who combines the disciplines of both geology and botany in one person, brings this unique equipment to bear upon the problem of the origin and evolution of flowering plants. Alfred E. Emerson ("The Evolution of Adaptation in Population Systems") considers different levels of population integration and the mechanisms involved in their adaptation. Ernst Mayr describes simply, clearly, and in an enlightening manner the processes by which evolutionary novelties become established. C. H. Waddington in "Evolutionary Adaptation" has contributed a sympathetic critique of evolutionary theory. On excellent grounds he offers the emendation that the evolutionary system must be considered to involve at least four major subsystems: (1) The *genetic system* embracing the whole chromosomal-genic mechanism of hereditary transmission; (2) *natural selection*; (3) the *exploitive system*, comprising the set of processes by which animals choose and often modify one particular habitat out of the range of environmental possibilities open to them; and (4) the *epigenetic system*, the sequence of causal processes which bring about the development of the fertilized zygote into the adult capable of reproduction. These subsystems are, of course, mutually interacting and interdependent. This is a conception of the evolutionary process very different from that which supposed that evolution was essentially random, unorganized, and operating through an essentially unconnected natural selection. Some day we may yet live to see nineteenth century concepts of "chance," "struggle," and "competition" as factors in evolution critically revalued. Waddington's contribution is a step in the right direction.

Th. Dobzhansky discusses the role that the environment plays in the evolutionary process. Environment produces the challenge, but it does not order the response that the organism makes to that challenge; it is natural selection that does that. But natural selection has no foresight. The changes that take place in the evolutionary process must be useful to their carriers in the environments in which they live at the time the changes occur. But, as Dobzhansky points out, even traits which are injurious when considered in isolation may really be adaptive, if they become established in evolution because they happened to be concomitants of useful traits. "Thus difficult childbirth—an absurdly

unadaptive trait—appears to be an adjunct of the erect body posture, which is clearly adaptive." This is an interesting point, but it is a question whether difficult childbirth is an adjunct of the erect posture. There is some evidence that the birth process is not unattended with difficulty in many non-erect animals. However that may be, is it not possible that difficult childbirth in the human species has its own adaptive value? The difficulty with which the human being is born emphasizes, as nothing else can, the preciousness as well as the precariousness of human life, and hence the potential adaptive value of labor and difficult childbirth in inducing those attitudes in adults which will increase the chances for survival of the baby once it is born.

Dobzhansky's definition of evolution as "a creative response of living matter to environmental opportunity" is surely a most nutritious cud to chew on.

Sewall Wright in "Physiological Genetics, Ecology of Populations, and Natural Selection," considers the mathematical framework of the theory of evolution at a succession of levels of complexity. This is a fundamental contribution, as are the conclusions to which it leads. Since, as Wright so charmingly puts it, there is no formula for serendipity, and evolution is an ever continuing process which is essentially unpredictable in its details, the mathematical theory developed in these pages by Wright is deterministic only in an exceedingly limited sense. Nevertheless it is an exceedingly illuminating and pragmatically valuable one.

Alexander J. Nicholson provides a stimulating discussion of the influence of population dynamics upon natural selection. He clarifies a great many processes which are only vaguely understood by many biologists, and draws attention to the much-neglected principle of compensatory reaction, the reaction whereby populations are adjusted to cope with changes in their environment by modifying their densities. This is a principle which has undoubtedly played a considerable role in the history of man's evolution, and it is one which has been severely neglected in discussions of man's evolution. It is obvious that during periods of famine there are many reactive responses possible by which the density of the population is reduced, not simply by starvation of the weaker, but by an actual reduction in fertility rates and an increase in malformation rates. Dr. Nicholson does not deal with these matters. I wish someone would. Nicholson's paper is full of nourishment, and it requires prolonged and repeated chewing in order to achieve the most beneficial results. It is very much worth the effort entailed. In "Morphology, Paleontology, and Evolution," Everett C. Olson offers some

very pertinent criticism of some aspects of contemporary evolutionary theory, while Marston Bates reflects interestingly on the relationships between ecology and evolution. In "Comparative Physiology in Relation to Evolutionary Theory" C. Ladd Prosser has written an exemplary and stimulating study stressing the importance of non-morphological, functional traits in the discussion not alone of the evolutionary process but also in the matter of taxonomy. Physiological adaptations of various kinds within the organism are no less important than physiological adaptations between the organisms and their environments. Natural selection applies to cellular processes as well as to more macroscopic organisms. Professor Prosser sees as the next area of breakthrough in the field of biology our learning how environmental stresses interact with cellular components to initiate the sequence of adaptive reactions. *That* will, indeed, be an exciting achievement.

In "Behavior, Systematics, and Natural Selection," N. Tinbergen discusses the extent to which taxonomic characters must be assumed to be due to natural selection. For this purpose Tinbergen utilizes the behavior characters mainly of birds—a difficult attempt most interestingly carried out. G. F. Gause in "Darwinism, Microbiology, and Cancer," presents a good discussion of the respiratory-damage theory of the causation of cancer, and with this paper Volume I is concluded.

The Evolution of Man: Mind, Culture, and Society. $10.00.

Volume 2 opens with a paper on "Evolution, History, and Culture," by A. L. Kroeber. This brilliant study of the historical development of anthropology reminds us of the loss that the historical sciences, and anthropology in particular, has suffered in the death of this great man. It is a contribution far too difficult to summarize in a few words, and rather than attempt it, I would urge it upon the attention of every interested reader.

L. S. B. Leakey, in "The Origin of the Genus *Homo*," gives a valuable account of the relevance of the discovery of African fossil hominoids, with the most important of which he was associated, for our understanding of the origin of man. His account of *Zinjanthropus boisei* is especially interesting.

S. L. Washburn and F. C. Howell in "Human Evolution and Culture" give an excellent account of the evolution of man, except when it comes to their stand on the advent of *Homo sapiens*. Here they bracket Swanscombe man with Steinheim man, and hence convert the former into *ein Halbneanderthaler*. Their handling of Fontéchevade man would, however, put a whole coda of Philadelphia lawyers to shame! Here is

what they write—in a footnote—"Vallois has clearly demonstrated, from very fragmentary evidence, the extent to which this form [Fontéchevade] differed from contemporaneous early Neanderthal peoples, with which such a form apparently coexisted. However, it seems certain that the prevalent and presumably dominant human form during the earlier Upper Pleistocene was an essentially primitive type with large brow ridges and other very distinctive morphological features." (p. 44). The crucial point is that what Vallois "clearly demonstrated" was that Fontéchevade was almost certainly a *sapiens*, and furthermore the archeological evidence is also clear that Fontéchevade man *preceded* Neanderthal man. If this is so, and it *is* so, what becomes of the authors' theory that *Homo sapiens* is a recent upstart? For the rest, the article is excellent, and the authors' point that "tool-using, ground-living, hunting" as "a way of life created the large human brain rather than a large-brained man discovering certain new ways of life" is being increasingly supported by recent analyses of the evidence.

Cesare Emiliani in "Dating Human Evolution" discusses the new methods with which he has been associated, and Edgar Anderson reviews the evidence on the domestication of plants. Stuart Piggot offers a stimulating series of ideas on "Prehistory and Evolutionary Theory," and unfortunately makes Erasmus Darwin the father of Charles. He was, of course, his grandfather. F. H. Bordes contributes a fundamental paper on "Evolution in Paleolithic Cultures" which should be required reading for all interested in this period. Gordon R. Willey has written a valuable analysis of the evolution of culture in the native New World, and concludes that this has occurred in an essentially independent manner. In "Levels in Prehistory: A Model For the Consideration of the Evidence," R. J. Braidwood proposes a new classification and nomenclature of cultural levels in prehistoric times which makes good sense, and in "The Evolutionary Process in Early Civilizations," R. M. Adams makes some excellent points on the adaptive characters of the growth of civilizations.

H. W. Magoun traces the development of concepts of brain function under the influence of Darwinian evolutionary theory, and incidentally gives an interesting account of the relations of Darwin and Herbert Spencer. What a melancholy, and wholly undeserved fate, Spencer has suffered! His fame has been totally eclipsed by that of Darwin. Someone ought to make available what is worth reading of Spencer, in a portable volume, for there is much of him that is still very much worthwhile.

Alexander von Muralt's "A Decisive Step in Evolution: Saltatory Conduction," is among the most fascinating of all the papers contributed

to the centennial. What it lacks in length it makes up in substance. It presents beautifully the reason for the improvement in the construction, economy, and performance of the peripheral nervous system: "saltatory" conduction in myelinated nerves is from node to node with resulting conduction velocities exceeding 100 m/sec in warm-blooded animals. Per gram of nerve, the myelinated nerve is three to four times more efficient in oxygen consumption than the unmyelinated nerve.

In "Pavlov and Darwin" W. Horsley Gantt has written a fascinating account of the points of contact between Darwin and Pavlov, and of the work of the latter, and its development by Gantt and others.

Leslie A. White in "The Evolution of Minding" discusses four stages of behavioral interaction of the organism with things, and once again cleverly distinguishes between signs and symbols. Man has a virtually unique capacity for the latter.

In "Becoming: The Residue of Change" R. W. Gerard defies resuming in a sentence. It is full of stimulating passages, and worth reading several times over; merely to say that it deals illuminatingly with the evolution of change from lower to higher levels of integration would be to do it an injustice.

Ernest R. Hilgard examines the development of psychology under the influence of Darwin, and in "The Evolution of Man's Capacity for Language" Macdonald Critchley discourses on the origin of language. He miscredits L. S. Palmer with being a dentist, when he is in fact a physicist, and his article would have been more useful had it been accompanied by a bibliography.

A. Irving Hallowell in "Self, Society, and Culture in Phylogenetic Perspective," has written a contribution of fundamental importance, in which he brilliantly considers the evolution of man as a human being. In this paper Hallowell raises so many questions that the attempt to discover the answers to them would keep a whole regiment of investigators busy for the next generation.

Henry W. Brosin in "Evolution and Understanding Diseases of the Mind," presents an admirable survey of recent integrative advances in the study of mental illness.

Hermann J. Muller in "The Guidance of Human Evolution" presents a profound discussion of the means by which human beings can more intelligently and effectively take their own future into their own hands.

Sir Charles Galton Darwin in "Can Man Control his Numbers" deals with the central problem of the immediate future, overpopulation. He realistically sets out the consequences which will follow if this problem

is not adequately dealt with at once, and since it will not be adequately dealt with in time, the probabilities are high that by the end of this century the world will be populated by 5 billion people. Fifty years hence it will be too late to reduce that number significantly. Thus ends volume two, somberly and seriously challenging.

Issues In Evolution. $7.50.

Volume 3 begins with an account of "Creation and Evolution in the Far East," by Ilza Veith. Among other things, it is interesting to discover that in the 19th century Darwinism made its way into China principally through the writings of T. H. Huxley, and that very soon social Darwinism had developed as a self-justificatory device for precisely the same reasons as it had done in the western world.

Quite frankly J. Franklin Ewing's "Current Roman Catholic Thought on Evolution" affords ground for believing that what may pass for "thought" on the subject of evolution among Catholic thinkers, has not reached, shall we say, a very high level of sophistication?

Jaroslav Pelikan's "Creation and Causality in the History of Christian Thought" is a theologian's account, and it is interesting.

But for sheer delight the "'At Random' A Television Preview" in which Julian Huxley, Charles Galton Darwin, Harlow Shapley, Adlai Stevenson, and Sol Tax appear on Kupcinet's evening program, is equalled by nothing in these fascinating volumes. Huxley shines with his accustomed brilliance, and Adlai Stevenson makes a good *proxime accessit.* Mainly it is population which is discussed, but there are good comments on evolution.

This review is already much too long, and it must be concluded. There are five admirable panels reported in detail, and many worthwhile things are said in all of them. Julian Huxley's convocation address "The Evolutionary Vision," is excellent, as one might expect. There is a television postscript in which some of the participants tell what the meetings have meant to them, Sol Tax tells how the idea for the Centennial came to him and how it developed, and there is a final program of pictures of the proceedings. This is followed by a satisfactory index to the three volumes.

Altogether these three volumes are a treasure. Students of the natural and behavioral sciences with their wits about them will mine them for years. Everyone who had anything to do with the making possible of these volumes deserves at least four cheers.

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BOOK NOTICES

CONTROL OF MALNUTRITION IN MAN. Prepared by the Subcommittee on Control of Nutritional Diseases. vi + 140 pp. American Public Health Association, New York, 1960. $1.50. This manual is a concise, authoritative presentation of current knowledge about the practical control of the various forms of human malnutrition (protein and calorie malnutrition, vitamin deficiencies, mineral deficiencies, and "other factors," such as anemias and phenylpyruvic oligophrenia).

MAN IN NATURE. By Marston Bates. xi + 116. Prentice-Hall, Englewood Cliffs, New Jersey, 1961. $1.50. A publication in the "Foundations of Modern Biology" series. It contains an introductory presentation of some of the major aspects of human biology (Primates, Human Evolution, The Varieties of Man, Human Populations, Health and Disease), plus 3 chapters on man's relation to the biosphere (Domestication, Agricultural Biology, Ecology and Economics).

BIOLOGICAL CLOCKS. COLD SPRING HARBOR SYMPOSIA ON QUANTITATIVE BIOLOGY, Vol. 25. xiii + 524 pp. The Biological Laboratory, Cold Spring Harbor, L. I., New York, 1960. Price not indicated. As could be expected, the majority of studies on biological rhythms and chronometry, reported in this volume, deals with plants and lower animals. Yet man is not altogether forgotten. In the section on "Circadian Rhythms in Animal Physiology," Theodore Heilbrügge presents observations on the development of the diurnal rhythm of various functions (pulse rate, body temperature, sleep and wakefulness, electrical skin resistance, kidney functions) in infants; the monophasic day-night rhythm develops out of polyphasic cycles. Mary C. Lobran describes studies of 24-hr. patterns of urinary excretion in the adult man. Diurnal fluctuations in body temperature were also considered. Both laboratory experiments, involving shift to shorter (21 hrs.) and longer (27 hrs.) "days," and field observations in the Arctic were carried out. The author concludes that environmental stimuli, especially occurrence of a period of daylight which coincides with the normal hours of activity, are needed "to bring about the full expression of his 24-hr. physiological rhythms" (p. 331).

STRUCTURE AND FUNCTION OF MUSCLE. Edited by G. H. Bourne. Vol. II. *Biochemistry and Physiology*, xiii + 593 pp.; Vol. III. *Pharmacology and Disease*. xiv + 489 pp. Academic Press, New York, 1960 and 1961. $16.50 and $15.00. These volumes complete the encyclopedic reference work. The second volume contains, among a multiplicity of other topics, chapters on the physiology of muscular exercise and on fatigue of the working individual. In the 3rd volume the chapters of special relevance to human biology are those dealing with the effect of nutritional deficiencies, age changes, and the genetic aspects of muscular and neuromuscular diseases.

RECENT ADVANCES IN HUMAN NUTRITION. By J. F. Brock. xii + 454. Little, Brown and Co., Boston, Massachusetts, 1961. $11.50. The volume is directed primarily to specialists in clinical medicine but it will be found useful by all interested in the polymorphous field of human nutrition. The editor has provided a readable general, systematic review of advances in our knowledge of nutrients, dietary requirements, and dietary deficiencies. Of special interest is the chapter on "Long-term cumulative effects of malnutrition and customary dietary patterns" (pp. 132-140) in which consideration is given to such topics as diet and degenerative diseases, obesity, growth (stature), and longevity. Part II of the volume consists of 15 chapters by some 20 authors on special subjects. These include trends in nutrition research in Latin American countries (food habits, assessment of nutritional status, endemic goiter) and effects of altered nutrition on the skeletal system.

MINERAL METABOLISM. Edited by C. L. Comar and Felix Bronner. xv + 879. Academic Press, New York, 1961. $14.50. In the second part (Part B) of Vol. 1 (Principles, Processes, and Systems) of this comprehensive treatise especially relevant are the sections on the structure and chemistry of bone, on minerals in nonosseous connective tissues (including cartilage), and on the hard dental tissues.

RACE AND SCIENCE. 506 pp. Columbia University Press, New York, 1961. $5.00 The volume contains 11 essays, written at the initiative of UNESCO and published separately, over the last 10 years, in the form of brochures in the series "The Race Question in Modern Science." They include, among others, Juan Comas' "Racial Myths," Michel Leiris' "Race and Culture," Claude Lévi-Strauss' "Race and History," L. C. Dunn's "Race and Biology," G. M. Morant's "The Significance of Racial Differences," Harry L. Shapiro's "Race Mixture," and Otto Klineberg's "Race and Psychology." The two UNESCO statements on race (the original, exuberant 1950 version and the revised, toned-down 1951 version prepared by a group of physical anthropologists and geneticists) are given in the appendix.

BECOMING MORE CIVILIZED. By Leonard W. Doob. xii + 33 pp. Yale University Press, New Haven, Connecticut, 1960. $6.00. This intriguing psychological exploration is focused "not upon whether less civilized men learn to smoke cigarettes or to put on pants but what changes occur in their way of thinking, and in their personalities." The novelty is partly that given by the area of field work (Luo, Ganda, Zulu in Africa; Jamaica), partly by the questions asked, but most importantly, perhaps, by the determined effort at quantification of the observations. "Stress is placed upon quantitative data, no matter how trivial or even statistically unconvincing, not because they lead rapidly to wisdom but because they suggest the need for precision if better truths are to be discovered." In the present reviewer's opinion, this is one of the best samples of 'psychological anthropology' on the record.

PSYCHOPHYSIOLOGICAL ASPECTS OF SPACE FLIGHT. Edited by Bernard E. Flaherty. x + 393 pp. Columbia University Press, New York, 1961. $10.00. The frame of reference was provided for the symposium by the point of view "that man's behavior is a joint function of many kinds of influences arising both inside and outside the body, and that the understanding and control of this behavior can be achieved only through the joint effort of all the life sciences." Consequently, "the conference agenda embraced an unusual variety of topics ranging from the psychosocial problems of small groups, through the medical and psychological problems of selection, training, and maintenance to the bedrock problems of neurophysiology and endocrinology."

HUMAN NUTRITION, HISTORIC AND SCIENTIFIC. Edited by Iago Galdston. xvii + 321 pp. International Universities Press, New York, 1960. $6.00. Some are still apt to think of the science of nutrition as a fairly thin slice of biochemistry. The present volume should help to establish nutrition as a broad field of inquiry, a field with a long past, but a short history ("Eating as an art is very old, but as a science, very young"; C. G. King), with roots in agriculture, core in research on metabolism, and branches extending from behavioral sciences (specifically, cultural anthropology and the study of food habits), to demography (Fairfield Osborn's chapter on "Nutrition and Population Pressure") and public health.

TEXTBOOK OF MEDICAL PHYSIOLOGY. By Arthur E. Guyton. xxvi + 1181 pp. Saunders, Philadelphia, 1961. $15.50. The second, thoroughly revised (but, fortunately, not much enlarged) edition of an outstanding, single-author textbook. The functions of the human body are presented as interrelated activities of different organ systems going on within a single organism, controlled by a multiplicity of regulatory mechanisms, with emphasis on the automaticity of the life process.

MODERN FACTOR ANALYSIS. By Harry H. Harman. xvi + 469 pp. University of Chicago Press, Chicago, 1960. $10.00. Factor analysis had its origin as a technique for reducing interindividual differences manifested in a multiplicity of scores on "mental tests" to a workable number of more basic dimensions. In the author's appraisal, "the new computing techniques (largely made possible by the advent of electronic computers), the objective procedures for determining simple-structure solutions, and the statistical tests of hypotheses are largely responsible for bringing modern factor analysis out of the abyss of a psychological fetish to the heights of a respected branch of statistical multivariate analysis."

THE HAZARDS TO MAN OF NUCLEAR AND ALLIED RADIATIONS. vii + 154 pp. Her Majesty's Stationery Office, London, 1960. $1.35. The resumption, in the Fall of 1961, by the Soviet Union of test explosions in air of nuclear devices of unprecedented magnitude, makes very timely the present appraisal (and the parallel reports issued by U. S., British, and U. N. agencies, listed on p. 3). It is distressing to contemplate the present and future

trends against the optimistic outlook of this report, typified by the statement that "in the absence of further weapon tests, the figures for strontium 90 can be expected to start declining after an interval of a year or so" (p. 49). The report consists of 2 parts: The text proper, and the more technical appendixes. The latter include a chapter entitled "Recent Information on the Genetical Effects of Radiation: A Bibliography and Notes" (p. 59-65).

ADVANCES IN PSYCHOSOMATIC MEDICINE. Edited by A. Jores and B. Stokvis. 328 pp. S. Karger, Basel (Switzerland) and New York, 1960. sFr. 58.— In the first volume of the *Advances*, conceived as a series of monographs to be published at irregular intervals, of special interest is the section on "Concept of Constitution in Psychosomatic Medicine" (pp. 101-165), concerned with the correlation of "psychosomatic basic structure" (A. Jores) and disease. L. Rees ("Constitution and Neurosis," pp. 106-120) contributed a useful review of the present status of constitutional research. He notes that "morphological attributes which show marked variability between individuals and relative constancy within the individual are more likely to prove useful for human constitutional research."

CONCEPTS OF MEDICINE. Edited by Brandon Lush. x + 286 pp. Pergamon Press, New York, 1961. $8.50. This collection of essays, the compilation of which was motivated by the editor's conviction that "knowledge advances by questioning established concepts and by framing new ones," covers a broad range of topics. Among these are H. Himsworth's plea for developing a new type of specialists—specialists whose function is integration of neighboring disciplines, counteracting the inherent and inevitable tendencies of modern science to isolation; W. B. Bean's critique of criticism in medicine and in biological sciences; Vannevar Bush's address on professional collaboration; a reprint of an older (1947) paper by John A. Ryle on "The Meaning of Normal"; H. C. Hoops' discussion "On the Philosophy of Research"; and C. N. H. Long's presentation on "The Selection, Care, and Preservation of Research Scientists."

MAN, RACE AND DARWIN. With an introduction and epilogue by Philip Mason. vi + 151 pp. Oxford University Press, New York, 1960. $2.90. The 11 papers constituting the symposium were presented at a meeting organized by the Royal Anthropological Institute and the Institute of Race Relations (an unofficial body established in 1958 to encourage and facilitate the study of the relations between races). In the present context only the scientific, biologically oriented contributions will be noted: Lancelot Hogben's "The Race Concept," N. A. Barnicot's "From Darwin to Mendel," G. A. Harrison's "The Biological Effects of Miscegenation," J. A. Fraser Robert's "A Genticist's View of Human Variability," and Philip E. Vernon's "Race and Intelligence."

EDEMA: MECHANISMS AND MANAGEMENT. Edited by John H. Moyer and Morton Fuchs. xxix + 833 pp. Saunders, Philadelphia, 1960. $15.00. Excessive

water retention is a part of a variety of clinical syndromes, including the "hunger edema." Non-medical human biologists will be interested in the data on the content of water and electrolytes (sodium, potassium, chloride) in muscle, skin, and subcutaneous adipose tissue (p. 454), and in the chapter by G. R. Menely et al. on "Factors which Influence the Amount and Distribution of Total Body Sodium and Potassium" (pp. 145-153). Reference is made to the promising technique of determining total body potassium in the living man by measuring the gamma rays emitted by the naturally occurring radioisotope K^{40}. Since potassium-40 occurs in a constant ratio to the nonradioactive potassium, it can be used as a natural tracer and serve as indicator of the "lean oxidizing protoplasmic mass."

CLASSICS IN BIOLOGY. xxx + 351 pp. Philosophical Library, New York, 1960. Price not indicated. These days and years in which the demands of scientific specialization tend to restrict one's reading to a narrower and narrower segment of the technical literature, it is a pleasure to browse on occasion, "out of bounds" and in a lighter mood, through the green pastures of the life sciences and to converse with great men about some of the greater problems of biology. The present collection, to which an Introductory Reading Guide was prepared by Sir S. Zuckerman, will serve well such a purpose.

MEDICAL AND BIOLOGICAL RESEARCH IN ISRAEL. Edited by Moshe Prywes. xx + 562 pp. Grune and Stratton, New York, 1960. No price indicated. In this comprehensive, well-documented survey especially relevant are the chapters on demography and social structure, epidemiology of noninfectious diseases, nutritional problems, and genetics of the population (prenatal diagnosis of sex by cytological methods, prenatal identification of blood groups, sex-chromosome constitution and human sexual development). Brief reference to human genetics is made also in the wider context of genetics research (p. 283).

HUMAN ECOLOGY *and* HEALTH. By Edward S. Rogers. xvi + 334 pp. Macmillan, New York, 1960. $7.75. The title of the preliminary editions (*Background for the Study of Public Health and Medical Administration*) identifies the book more precisely. It is an outgrowth of years of teaching graduate students in public health. Part I deals with the demographic background, Part II describes the health status of the population of the United States, Part III develops "An Ecological Approach to the Concept of Health and Illness," with emphasis on the importance of multiple and complex environmental influences in disease causation. The term "ecologic," as used by the author, includes the relationship between man and his *total* environmental.

HUMAN FACTORS IN JET AND SPACE TRAVEL. Edited by S. B. Sells and Charles A. Berry. xvi + 386 pp. Ronald Press, New York, 1961. $12.00. The book has been prepared to provide up-to-date information, in systematic

form, on the human problems of high-speed, high-altitude flight. Its content reflects the conviction that, in regard to many of the biological problems, there is no sharp dividing line between atmospheric flight and space travel. It reflects, also, the fact that human-factors research and development has involved the joint effort of many disciplines, including physiology, psychology, medicine, and engineering. A detailed index adds to the usefulness of the volume which consists of 13 technical chapters and Hamilton B. Webb's "Speculations on Space and Human Destiny."

PERFORMANCE CAPACITY—A SYMPOSIUM. Edited by Harry S. Spector, Josef Brožek, and Martin S. Peterson. Sponsored by the Advisory Board on Quartermaster Research and Development, Committee on Foods, National Academy of Sciences—National Research Council, Washington, D. C. xi + 257 pp. Quartermaster Food and Container Institute, 1819 W. Pershing Road, Chicago 9, Illinois, 1961. Available free of charge. The aim of the symposium, interdisciplinary in character, was to review and evaluate methods (biochemical, physiological, behavioral) for the assessment of performance capacity. Consideration was given to their applicability to the study of stresses (physical work, heat, cold, restricted food intake, dehydration, ionizing radiation, emotional stress), both under laboratory and field conditions, in man and in experimental animals. A review of recent publications bearing upon the problems discussed at the conference, held in 1957, is provided in the form of an epilogue.

THE METABOLIC BASIS OF INHERITED DISEASE. Edited by John B. Stanbury, James B. Wyngaarden, and Donald S. Fredrickson. xiv + 1, 477 pp. McGraw-Hill, New York, 1960. $30.00. This is "magnum opus," in external size (and cost) as well as in the competence and thoroughness in documenting the astonishing growth of human biochemical genetics that had taken place since Sir Archibald Garrod's studies on alcaptonuria and his monograph on "Inborn Errors of Metabolism" (1923), mostly in the last two decades. As the reader is reminded by the editors in the introductory chapter on "Inherited Variation and Metabolic Abnormality," it was Garrod who "developed the concept that certain diseases of lifelong duration arise because an enzyme governing a single metabolic step is reduced in activity or missing altogether." The principle "one gene, one enzyme" was formulated by G. W. Beadle (1945). The diseases with inherited metabolic basis are presented in 10 sections, dealing with the substances that are involved (carbohydrates, amino acids, lipids, steroids, purines and pyrimidines, metals, porphyrins), tissues (blood and blood-forming tissues), functions (diseases manifest primarily in renal tubular transport), and deficiency of circulating enzymes or plasma proteins.

PRINCIPLES OF HUMAN GENETICS. By Curt Stern. x + 753 pp. Freeman, San Francisco, 1960. $9.50. The second edition of this first-class textbook, revised and expanded, takes full account of the manifold advances made in the decade that elapsed since the publication of the first edition (1949).

Its aim remains the same: "the presentation of the principles of human genetics as a means of understanding some of the diversity, as well as the underlying community, of mankind." Yet the more "applied" aspects (medicolegal applications of genetics, relationship of heredity to disease, genetic hazards of radiation, genetics of behavior) are also considered.

THE ETHICAL ANIMAL. By C. H. Waddington. 230 pp. Allen and Unwin, London, 1960. 25s. The author, professor of animal genetics at the University of Edinburgh, has been concerned for some 2 decades with the challenging theme of "Science and Ethics." Here he spells out the arguments for his thesis that ethical beliefs (beliefs about the nature of the most authoritative demands) play an essential role in enabling the human race to continue evolving in the direction which in general terms continues that of animal anagenesis (i.e., progressive evolution, improvement), but by a mechanism which is peculiarly characteristic of *homo sapiens*: effective transmission of information from one generation to the next through social teaching and learning. In man, "heredity" operates through a socio-genetic system of transmission. Examination of the direction of evolution (of anagenesis) provides the criteria and the framework within which our ethical beliefs can be evaluated.

ENDEMIC GOITER. Monogr. Series No. 44, World Health Organization, Geneva (Switzerland). 471 pp. 1960. $8.00. In many countries, endemic goiter continues to persist as a threat to the physical, social and economic well-being of individuals and communities. The present publication offers a comprehensive discussion of history, etiology, physiology and pathology, prevalence and geographical distribution, as well as health significance, techniques of goiter surveys, and the therapy and iodine prophylaxis of endemic goiter.

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ERRATA

HUMAN BIOLOGY, VOLUME 33, NUMBER 2, PAGE 246:

"DISTRIBUTION AND INHERITANCE OF MIDDLE PHALANGEAL HAIR IN A WHITE POPULATION OF SÃO PAULO, BRAZIL"

BY P. H. SALDANHA AND SONIA GUINSBURG

In editing of table 8, items of columns 4, 5 and 6 of the original manuscript were uncorrectly changed. Table 8 must be read as below:

TABLE 8

Frequencies of types of sib pairs observed and expected from monogenic recessivity and random combination hypothesis (A indicates presence and B absence of middle phanangeal hair in both hands)

TYPE OF SIB-PAIR COMBINATION	OBSERVED FREQUENCY	EXPECTED FREQUENCY				
		RECESSIVITY HYPOTHESIS	χ^2 (d.f. = 1)	P	RANDOM COMBINATION HYPOTHESIS	χ^2 (d.f. = 2) P
AA	11	13.8	0.57		8.6	0.67
AB	18	12.3	2.64		23.0	1.09
BB	18	20.9	0.40		15.4	0.44
Total	47	47.0	3.61;	> 0.05	47.0	2.20; = 0.35
		A = 42.6%	$\bar{B}$ = 57.4%		p = 0.242	

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a record of research

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